The Future of Human Health, Longevity, and Health Costs[*]

Sebastian Böhm[†], Volker Grossmann[‡] and Holger Strulik[§]

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Abstract

This paper investigates the future of human longevity, morbidity and health costs in a novel, multi-period overlapping generations model with endogenous medical R&D and endogenous survival that is closely associated with morbidity. We capture biologically founded ageing based on gerontology research in order to calibrate our model for the UK. The baseline policy scenario of health care access suggests substantial future increases in human longevity that are associated with both reductions in morbidity and a rising health expenditure share in GDP. Stabilizing the health expenditure share by rationing health care has potentially sizable effects on morbidity and longevity in the longer run, associated with reduced medical R&D incentives. The implied welfare effects may be substantially negative particularly for future generations.

Key words: Longevity; Medical R&D; Morbidity; Health Care; Rationing.
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1 Introduction

A salient feature of structural economic development over the last decades is the secular expansion of both the health sector and human longevity. In the U.S., health expenditure per capita grew by on average 4.1% annually since 1970 to a level of about 18% of GDP four decades later (Chernew and Newhouse, 2012; Gaynor et al., 2015). Starting at lower levels, other developed countries experienced similar rates of increase of the health sector such that, across the board, health expenditure increased faster than GDP.

Scholars agree that both the rise of health expenditure and improvements in longevity are related to medical technological progress.[1] Recent examples of health innovations include computerized diagnostic tests (e.g. for medical imaging), personalized cancer therapy, and new treatments of virus infections like HIV or Hepatitis C. More generally, Lichtenberg (2007) shows that later vintages of pharmaceuticals are more powerful in the reduction of health deficits. Considering the evolution of 92 potentially lethal diseases he finds that conditions experiencing greater pharmaceutical innovation tend to have greater declines in mortality rates.[2]

Consistent with such evidence, this paper proposes a new approach to study the interdependence of medical R&D, health expenditure, longevity and the health status of an age-structured population. We develop a a multi-period overlapping generations model where endogenous medical progress affects morbidity in interaction with access to health goods. In turn, morbidity affects mortality rates.

We employ the *health deficit index* developed by gerontologists (Mitnitski et al., 2000,

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[1]As argued convincingly by Chernew and Newhouse (2012), the *persistent* increase of health expenditure shares requires at least one other persistently growing explanatory variable (and thus rules out institutional changes like health care reforms and other only occasionally changing variables). Okunade and Murthy (2002) establish a long-run relationship between medical R&D expenditure and health care expenditure. There may be a role for income as a driver of health costs, although some recent studies refute the luxury good hypothesis of health care by estimating an income elasticity of health expenditure below unity (Acemoglu, Finkelstein and Notowidigdo, 2013; Baltagi et al., 2016).

[2]A promising example of a potentially powerful future technology is "targeted genome editing" like the *clustered, regularly interspaced, short palindromic repeat* (CRISPR) technology. It gives rise to the development of novel molecular therapeutics for human disease. Also see The Economist (2016) for an overview on recent developments in anti-ageing research.

2002) and subsequently used in countless empirical studies in the natural sciences for measuring health status and its relation to mortality in a biologically founded way. The approach has two advantages. First, it enables us to calibrate our model. In contrast to health capital (a latent variable popular among economists; Grossman, 1972), health deficits are observed and easily quantifiable. In our model, in line with the conceptualization of morbidity and physiological ageing in gerontology research, individuals accumulate health deficits over the life-cycle which in turn determine mortality rates at a given age. The individual accumulation process of health deficits depends on the interaction between the extent to which individuals are provided with health goods to treat their illnesses and their available quality, that is endogenously determined by vertical R&D. Second, the approach enables us to understand the potential effects of changes in the access to health care by putting the empirically established path-dependency of health deficits at the center of the analysis. It implies that improperly treated health deficits lead to new ones that overall may considerably shorten life-time.

We apply the approach to make inferences about the future development of life expectancy and health expenditure, conditional on the extent of future access to health care. Consistent with the established importance of medical progress for health costs and human longevity, our calibrated model suggests substantial future gains in life expectancy that are associated with significant declines in morbidity. In our baseline scenario, these developments are related to an increase in the health expenditure share in GDP by about two percentage points until 2080.

Despite the good news on human health, the entailed increasing utilization of medical goods and services has already in the past raised concerns about fiscal sustainability of health insurance systems and, more generally, the overall desirability of such trends. It motivated the discussion of rationing access to health care as potential remedy to curb further rising expenditure shares (Aaron and Schwartz, 1990; Ham and Glenn, 2003; Singer, 2009). Indeed, health care rationing has become more and more visible in developed countries. For instance, the National Health Service (NHS) − managing

tax-financed health care with guaranteed access in the UK − rations hip replacements and knee surgeries (Edwards, Crump and Dayan, 2015; OECD, 2015) and severely limits coverage of a novel (albeit expensive) drug that for the first time heals Hepatitis C.[3]

Applying our framework to address this important debate suggests that rationing care in order to constrain health expenditure growth has severe "side-effects" on future health and longevity. Aside from the obviously detrimental effects on health of the current population it also reduces market size for new medical products, in turn suppressing medical R&D. We argue that, consequently, preventing an increase in the health expenditure share would, for instance, reduce remaining life-expectancy of an individual who has reached age 65 in year 2050 by almost 4 years.

This leaves us with the fundamental normative question how to decide on the trade-off between promoting longevity and limiting increases in health costs. For this purpose we propose a welfare analysis that compares different future scenarios of health care access. We assume that instantaneous utility of surviving individuals depend on their health status and material consumption. Marginal utility from consumption negatively depends on morbidity, in line with empirical evidence (Finkelstein, Luttmer and Notowidigdo, 2013). Our welfare analysis suggests that particularly future generations would incur dramatic welfare losses from rationing measures that stabilize the health expenditure share, despite increases in their disposable income associated with reduced health care spending. We estimate, for instance, that someone who is 20 years old in 2020 could

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[3]See http://www.hepatitisc.uw.edu/page/treatment/drugs/sofosbuvir-drug. NHS England has decided to provide treatment only to the 10,000 sickest persons of those being infected per year, a rather small fraction of the estimated 215,000 infected persons in the UK (https://www.theguardian.com/society/2016/jul/28/nhs-abandoning-thousands-by-rationing-hepatitis-c-drugs). Also other countries severely ration access to Hepatitis C treatment (World Health Organization, 2016) or discourage health spending in more general ways. For instance, in the mandatory German health system, if the amount of external costs attributable to a medical doctor exceeds a threshold per quarter, the doctor has to privately bear the costs above the cap. Contrary to many European health systems, US medicare (health insurance for the elderly) involves a co-insurance rate for pharmaceuticals of 25%. Co-insurance makes demand for pharmaceuticals price-elastic. In fact, in the US prices for pharmaceuticals are little regulated, compared to European health care systems. The fundamental issue of rationed health care provision is nevertheless present as well in the US, albeit in different form.

expect a welfare loss of 14-24 percent from the regime switch.[4] For a 20 year old in 2050 the welfare loss could be 34-48 percent that is associated with a reduction in remaining life expectancy by about 10 years.

The reminder of the paper is organized as follows. Section 2 discusses our contribution in view of related literature. The model is presented in section 3. Section 4 provides the positive analysis of the evolution of life expectancy and mobidity under different health expenditure policies. Section 5 presents a comparative welfare analysis of different policy scenarios. The last section concludes.

2   Related Literature

Our main contribution is to highlight the interaction between endogenous medical technological progress and longevity as a function of access to health care. Most empirical studies of the determinants of health expenditure estimate medical technological progress as a residual. The study by the European Union (2010), for example, regresses health expenditure against income, the population share above 65, and a time trend, and interprets the time trend (of on average 2 percent annually) as the rate of medical technological progress. Treating medical technological progress as a time trend, however, is problematic when predictions are made on long-run developments of population health and health expenditure that do not account for potential changes in health care policy. Implicitly these predictions assume that health care reforms do not affect medical progress. In our study we challenge this view by modeling endogenous medical innovation and endogenous population health for different policy scenarios. In particular, we show that limiting the rise in health expenditure has a detrimental effect on health R&D through a market size effect that is associated with declining health care utilization. We thus formalize an idea

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[4]We measure welfare changes from a regime switch in the health care system by an equivalent variation measure. We ask by how much we would have to multiply material consumption levels under the baseline health policy scenario such that the *ex ante* life-time utility of a member of a given generation is the same as after the policy regime switch that forces the health expenditure share to remain at its current level.

that goes back to Weisbrod (1991) who argues that the expansion of U.S. health care insurance has induced increasing health R&D and newly developed technologies that, in turn, have driven up health care utilization and costs.[5] We show that the resulting decreases in mortality rates (i.e. rising life expectancy) are associated with further increases in health costs, consistent with empirical evidence (e.g. Zweifel, Steinmann and Eugster, 2005; Bech et al., 2011; Breyer, Normann and Niebel, 2015).

Related studies investigated the interaction of health R&D in "reduced form" by either assuming a direct utility gain from the consumption of pharmaceuticals (as in Garber, Jones and Romer, 2006, and Grossmann, 2013) or by assuming a direct impact of health good consumption on the mortality rate of a representative individual (as in Jones, 2016a). Garber et al. (2006) investigate the interaction between medical R&D of a monopolist and the generosity of the health care system, measured by the degree of coinsurance payment of individuals. New generations of pharmaceuticals are assumed to directly raise utility of individuals with the respective disease. Neither health nor longevity are explicitly modeled. Prices of pharmaceuticals are set in private markets (approximating the US health care system). In this setup, lower co-payments lead to higher demand and higher markups charged by drug producing firms.[6] Consequently, profits of firms may exceed consumer surplus of patients such that, in this sense, there could be too much demand and too much medical R&D. In contrast to that paper, which highlights the problem of moral hazard when prices for pharmaceutical are set on markets, we assume that prices are regulated (approximating the British and German health care system, among others). We then focus on the interaction between health expenditure, medical R&D, morbidity and longevity of an age-structured population in a dynamic

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[5]Testing this hypothesis, Acemoglu et al. (2006) could not show that the introduction of Medicare (the "Social Security Act of 1965" that covered hospital and doctor expenses) increased pharmaceutical demand and pharmaceutical R&D. This finding is not surprising, however, since coverage of pharmaceuticals was not introduced until 2006. Extending the scope of analysis, Acemoglu and Linn (2004), showed large market size effects of the aging baby boomers on the development and market entry of new (age-specific) pharmaceuticals.

[6]In a similar vein, Grossmann (2013) relates co-insurance rates to medical R&D incentives. By examining an oligopolistic pharmaceutical market with endogenous firm entry, he also shows that entry deregulation may lead to more pharmaceutical R&D despite lowering profits of pharmaceutical firms.

macroeconomic model.

More recently, Jones (2016a) proposes a macroeconomic model with horizontal health innovations that affect longevity of a single cohort that privately buys health goods (similar to Grossman, 1972) with a trade-off to material consumption (featuring "love-of-variety" of consumers in both sectors). By investigating the optimal allocation of R&D effort directed towards innovations for health and non-health purposes, it is shown that under a mild condition non-health technological progress may optimally converge to zero growth such that the health expenditure share optimally converges to 100 percent. The study makes an important, eye-opening contribution in the debate whether there is too much health care expenditure and it paves the way for our research. Our study, however, focusses on different research questions and shifts from the single-agent view to a multi-period, overlapping generations model with an explicit health care system and a biologically founded relationship between morbidity and mortality. In particular, we investigate the effects of health care (rationing) on health and longevity of an age-structured population and account for the path-dependency of health deficits.

In a development context, higher life expectancy may positively affect per capita income (e.g. Cervellati and Sunde, 2011). In fact, investments in human capital or entrepreneurship may be fostered because the gains of economic activity is spread on a longer time horizon. In advanced economies, however, longevity is enjoyed by retirees. Thus, publicly financed policy interventions to promote health good provision and health R&D do not necessarily raise per capita income and consumption levels. Rather there is a fundamental trade-off between longevity and material well-being that we examine in our welfare analysis.

Our paper is also related to a strand of recent studies that utilized the health deficit approach to (re-)investigate the Preston curve (Dalgaard and Strulik, 2014), the role of adaptation for health behavior and health outcomes (Schuenemann, Strulik and Trimborn, 2015), the education gradient (Strulik, 2016), the historical evolution of retirement (Dalgaard and Strulik, 2017), and the optimal design of social welfare systems (Gross-

mann and Strulik, 2015).[7]

Finally, there is a large literature outside economics that attempts forecasting future life expectancy by estimating statistical time trends. For instance, as acknowledged by Kontis et al. (2017) in a widely received paper that accounts for model uncertainty with a Bayesian model averaging approach, their "key limitation [...] is the inability to account for [...] changes in the social, technological, and health systems determinants of health" (p. 8). In our economic approach, we endogenize health technology and calibrate health care utilization, comparing implications of different regimes of future health care policy.

3   The Model

Consider the following multi-period overlapping generations model in discrete time, indexed by t, in which individuals age by accumulating bodily impairments ("health deficits"). In line with the evidence on human ageing, on average, individual health deficits correlate exponentially with age and are a highly relevant determinant of the probability of death (e.g. Mitnitski and Rockwood, 2002a, 2002b, 2005). Health goods are provided via a tax-financed health care system without coinsurance, like in the UK and Germany. Improved quality of provided health goods slows down the ageing process.

Private firms decide competitively on medical R&D. Also the final good sector and factor markets are perfectly competitive, whereas health good providers charge mark-up prices. Mark-up factors can be thought of being determined by negotiations between health care representatives and health good suppliers, again, like in the UK and Germany. There exists a perfect private annuity market. For simplicity, we assume that there is an international capital market that fixes the real interest rate, $\bar{r}$.

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[7]Grossmann and Strulik (2015) investigate the interaction between increasing health expenditure, which promotes longevity, and a publicly financed pay-as-you-go pension system that is challenged by (endogenously) changing demography. They do not incorporate health R&D or a health good sector, however. Moreover, their analysis is confined to two periods of life (with endogenous lengths).

3.1 Households

Each period a new cohort is born. Mortality is cohort- and age-specific and determined by health status, that is measured by the fraction of the health deficits an individual suffers from out of a long list of potential impairments ("health deficit index"), ranging from mild deficits (reduced vision, incontinence) to near lethal ones (e.g. stroke).[8]

Formally, the probability $m_{v,t}$ of a member of cohort v to die between period t and $t+1$, conditional on having reached age $t-v \geq 0$, is increasing as a function of the health deficit index at that age, $d_{v,t} \in [0,1]$. There exists a threshold deficit state $d_{max} \in (0,1)$ such that no individual survives beyond that state. Moreover, there is a maximum life span (irrespective of health deficits), T. These properties are captured by the parsimonious specification

$$m_{v,t} = \begin{cases} \frac{1 - e^{-\frac{(d_{v,t})^\phi}{\sigma}}}{1 - e^{-\frac{(d_{max})^\phi}{\sigma}}} \equiv \tilde{m}(d_{v,t}) & \text{if } d_{v,t} < d_{max} \text{ and } t < v + T - 1 \\ 1 & \text{otherwise,} \end{cases} \tag{1}$$

where we assume $\sigma > 1$ and $\phi > 1$.[9] Note that $\tilde{m}(0) = 0$ and $\tilde{m}(d_{max}) = 1$. As will become apparent, specification (1) enables us to capture empirically observed survival rates reasonably well with a small set of parameters. By definition, the survival rates and conditional mortality rates are related by

$$S_{v,t} = S_{v,v} \prod_{u=v}^{t-1} (1 - m_{v,u}) \text{ for } t \geq v + 1, \tag{2}$$

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[8]According to Rockwood and Mitnitski (2007) and Searle et al. (2008), the exact choice of the set of potential deficits is not crucial, provided that the set is sufficiently large. We present a typical list of health deficits from Searle et al. (2008) that serves to compute the health deficit index (often called "frailty index") in the Online Appendix (Table A.1).

[9]In the Online Appendix (Figure A.1) we present an empirical foundation of the close connection between mortality rates and the health deficit index from three survey waves of Canadian cohorts aged 65+ (Mitnitski, Bao and Rockwood, 2006). The relationship is strictly convex. Less that 4% of the total population had a deficit index above 0.35, implying a very high probability of death above this value. According to (1), we have $\tilde{m}'' > 0$ if $\left(1 - (d_{max})^\phi / \sigma\right) \phi > 1$, which will hold in our calibrated model.

i.e., $m_{v,t} \equiv -(S_{v,t+1} - S_{v,t})/S_{v,t}$. The initial cohort size in period v is $S_{v,v}$.

Each individual works for R periods and inelastically supplies one unit of labor in working age (and no labor afterwards).[10] We thus implicitly assume that, conditional on survival, labor supply is independent of health status.[11] The total units of labor supplied to the economy in period t are given by $L_t = \sum_{u=t-R+1}^{t} S_{u,t}$.

Households have preferences over material consumption and health status. They choose the consumption path that maximizes expected life-time utility. Because the interest rate is fixed, saving decisions of households do not affect firm decisions. We thus first analyze the supply side and introduce life-time utility later to analyze welfare implications of our model.

3.2 Production

There is a standard numeraire goods sector, producing a standard final good, and a health sector.

3.2.1 Numeraire Good Sector

The final good is chosen as numeraire. It is produced under perfect competition according to

$$Y_t = (K_t^Y)^\alpha (A_t L_t^Y)^{1-\alpha}, \tag{3}$$

$\alpha \in (0,1)$, where K_t denotes the physical capital input in period t, L_t^Y is the amount of labor in the consumption goods sector, and A_t is a measure of non-health knowledge. Its level is initially given by $A_0 > 0$ and exogenously grows over time with constant rate $g > 0$. Physical capital depreciates at rate $\delta^K \geq 0$. Thus, the user cost per unit of capital is given

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[10]Allowing for a positive elasticity of labor supply with respect to net wages rather than assuming exogenous labor supply would be conflict with the evidence that hours worked have declined over a longer time horizon in many growing economies (e.g. Lee, McCann and Messenger, 2007).

[11]In fact, at the individual level, a decline in health status does not seem to have a large effect on labor supply (see e.g. Jaeckle and Himmler, 2010, as well as Hokayem, and Ziliak, 2014). Introducing age-dependent labor supply or varying the retirement age does not affect the main insights of our analysis.

by $\bar{r} + \delta^K$. It is equal to the marginal product of capital, $\bar{r} + \delta^K = \alpha(A_t L_t^Y / K_t)^{1-\alpha}$. The wage rate, w_t, equals the marginal product of labor, i.e. $w_t/A_t = (1-\alpha)(A_t L_t^Y / K_t^Y)^{-\alpha}$. Thus,

$$\frac{w_t}{A_t} = (1-\alpha)\left(\frac{\alpha}{\bar{r} + \delta^K}\right)^{\frac{\alpha}{1-\alpha}} \equiv \omega. \tag{4}$$

3.2.2 Health Sector

The health sector provides patentable health goods (and services) like pharmaceuticals to treat illnesses. Production of one dose of a health good requires $\chi > 0$ units of labor. Thus, marginal production costs in period t are χw_t.

There is a continuum of potential illnesses that are represented by the unit interval, indexed by $j \in [0,1]$. For each illness, there is a competitive R&D sector aiming to advance the treatment quality. A successful innovator provides a quality level that is by an amount $\gamma > 0$ higher than the quality of the previous vintage. An innovator is formally awarded an infinitely-lived patent. As will become apparent, however, patent holders will frequently be driven out of business by future innovators. The quality of the latest vintage of health good j available in period t is denoted by $q_t(j)$. The quality of all health goods (including older vintages) may deteriorate over time at rate $\delta^Q \in (0, \gamma)$. In the case of pharmaceuticals, depreciation of quality captures mutations of bacteria and viruses, with resistance of antibiotics being a prime example.

Denoting by $\mu_{t+1}(j)$ the probability of a successful innovation to treat illness j that can be commercialized in $t+1$, the quality of health good j thus evolves according to

$$q_{t+1}(j) = \begin{cases} (1-\delta^Q)q_t(j) + \gamma & \text{with probability } \mu_{t+1}(j), \\ (1-\delta^Q)q_t(j) & \text{otherwise.} \end{cases} \tag{5}$$

Hence, the expected quality of a health good targeted to illness j in period $t+1$, $\mathbb{E}[q_{t+1}(j)]$,

10

is given by

$$\mathbb{E}[q_{t+1}(j)] = \mu_{t+1}(j)\left[q_t(j)(1 - \delta^Q) + \gamma\right] + (1 - \mu_{t+1}(j))q_t(j)(1 - \delta^Q). \qquad (6)$$

Let $l_t(j)$ denote the amount of labor devoted by a representative R&D firm in health sector j at t and assume that the perceived probability of a successful innovation is proportional to it:

$$\tilde{\mu}_{t+1}(j) = \tilde{\xi}_t l_t(j), \text{ with } \tilde{\xi}_t \equiv \xi \cdot (L_t^Q)^{-\vartheta}, \qquad (7)$$

$\xi > 0$, $\vartheta \in (0, 1)$, where L_t^Q is the aggregate amount of health R&D labor in t. $\tilde{\xi}_t$ is taken as given in the decision of R&D firms and captures a negative R&D ("duplication") externality: $\vartheta > 0$ implies a wedge between the private and social return to R&D that may arise because firms do not take into account that rivals may work on the same idea such that, from a social point of view, some of the R&D is duplicated.[12] In a symmetric equilibrium, where $l_t(j) = L_t^Q$ for all $j \in [0, 1]$, we obtain $\tilde{\mu}_{t+1}(j) = \tilde{\mu}_{t+1} = \xi \cdot (L_t^Q)^{1-\vartheta}$ for all j.

There also may be innovations that occur unintentionally or are commercialized by non-profit innovators like public research institutions.[13] They become effective in $t + 1$ with probability $\bar{\mu}_{t+1}$. Let $Q_t \equiv \int_0^1 q_t(j)\mathrm{d}j$ denote the average quality of the latest vintages of health goods ("stock of medical knowledge"). We assume that there is an intertemporal spillover of the form

$$\bar{\mu}_{t+1} = \eta Q_t, \qquad (8)$$

$\eta \in [0, \delta^Q/\gamma)$. Thus, the total probability of medical progress in any sector is given by

$$\mu_{t+1} \equiv 1 - (1 - \bar{\mu}_{t+1})(1 - \tilde{\mu}_{t+1}) = \eta Q_t + (1 - \eta Q_t) \cdot \xi \cdot (L_t^Q)^{1-\vartheta}. \qquad (9)$$

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[12]For $\vartheta \to 1$, social returns to medical R&D investment approach zero. The argument is analogous to that in Jones and Williams (2000) for a non-health R&D context.

[13]The inventions of Penicillin and Viagra are prime examples of major breakthroughs that were not intended to treat the health problems they target today.

By the law of large numbers, there is no aggregate risk. Thus, $\int_0^1 \mathbb{E}[q_{t+1}(j)] \mathrm{d}j$ is deterministic and equal to Q_{t+1}. According to (6), it evolves as

$$Q_{t+1} = \gamma \mu_{t+1} + (1 - \delta^Q) Q_t, \tag{10}$$

where initial level $Q_0 > 0$ is given. Substituting (9) into (10), we obtain

$$\frac{Q_{t+1} - Q_t}{Q_t} = \frac{\gamma(1 - \bar{\mu}_{t+1})\tilde{\mu}_{t+1}}{Q_t} - \tilde{\delta}^Q = \frac{\gamma(1 - \eta Q_t)\xi(L_t^Q)^{1-\vartheta}}{Q_t} - \tilde{\delta}^Q, \tag{11}$$

$\tilde{\delta}^Q \equiv \delta^Q - \gamma \eta > 0$. Thus, the growth rate of Q is a declining function of its level, becoming negative without intentional R&D (i.e. $Q_{t+1} < Q_t$ if $L_t^Q = 0$).

3.3 Health Care Provision

In many advanced countries, the bulk of individuals exclusively rely on a highly regulated health system with compulsory contributions (e.g. Germany and Switzerland) or is tax-financed like the National Health Service (NHS) in the UK. For simplicity, we assume that health goods are exclusively provided by such health system which may also include measures to ration health care provision.

We do not consider the possibility of "out-of-pocket" health payments or coinsurance. Although the absence of these features are limitations of our analysis, we capture reasonably well the health system of the UK, to which we calibrate our model. The NHS, like in the mandatory German health system, does not demand copayments. Out-of-pocket health expenditures as fraction of total UK health expenditure have been around 10 percent in the 2000s (OECD, 2016). Also private health insurance coverage has been at a modest level (10.5 percent in the year 2014). Many salient health goods, like surgeries treating orthopedic deficits or drugs for treating cancer and virus infections, may indeed be unaffordable for the bulk of individuals (presumably those who do not have private insurance either), if not covered by NHS.

The price mark-up of health goods can be thought of as an outcome of negotiations between the health care provider and (a representative body of) health good producers like pharmaceutical companies.[14] For instance, in the UK, prices for pharmaceuticals are regulated and based on a non-contractual agreement between the UK Department of Health and the Association of the British Pharmaceutical Industry. Similarly, in Germany, health care suppliers negotiate with pharmaceutical companies the maximum price covered by the mandatory health insurance.

Suppose that prices for older vintages are bid down to marginal costs and are not supplied anymore, whereas the industry leader can charg a mark up that is increasing in his quality advantage vis-à-vis previous vintages. Denote by $\mathfrak{q} > 0$ the (absolute) quality advantage of the industry leader over the competitor with the second-highest quality product in the same market. We assume that the mark up factor is given by $1 + f(\mathfrak{q})$, where f is an increasing and strictly concave function that fulfills $f(0) = 0$. It captures the price setting power of health good providers as a function of its quality advantage in the market. If the leading firm is one step ahead of the closest competitor (i.e. $\mathfrak{q} = \gamma$), it gets a profit per unit sold that is equal to $f(\gamma)\chi w$. If the leading firm is two steps ahead of the closest competitor (i.e. $\mathfrak{q} = 2\gamma$), it gets a profit per unit equal to $f(2\gamma)\chi w$. The profit increase for the industry leader by innovating, i.e. becoming two steps rather than one step ahead, is $[f(2\gamma) - f(\gamma)]\chi w$. Since strict concavity of f and $f(0) = 0$ imply $f(2\gamma) < 2f(\gamma)$, we have $[f(2\gamma) - f(\gamma)]\chi w < f(\gamma)\chi w$. Thus, it does not pay off for the leader to innovate. The incumbent firm would strictly prefer to invest in R&D in a second market rather than advancing its latest vintage.[15] Consequently, the incumbent is driven out of business when there is an innovation in the market it leads and leader's quality advantage to the closest competitor is exactly $\mathfrak{q} = \gamma$. Hence, the price p_t of each health

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[14]Pharmaceutical companies may draw their negotiation power via lobbying and marketing that influences government negotiators and public opinion, respectively, on the merits of pharmaceuticals. For instance, interest groups representing the pharmaceutical sector strongly argue that they need to earn high profits enabling them to conduct R&D and therefore have to charge high prices that should be covered by health insurance.

[15]See Grossman and Helpman (1991) for a similar argument in a context of Bertrand competition.

good is given by

$$p_t = \Gamma \chi w_t = \Gamma \chi \omega A_t, \tag{12}$$

where $\Gamma \equiv 1 + f(\gamma)$ is the mark-up factor.

3.4 Health Deficit Accumulation

We assume that physiological ageing starts when individuals become economically active, i.e. consume and supply labor.[16] Modern gerontology describes ageing as an accumulation of health deficits (e.g. Mitnitski and Rockwood, 2002a, 2002b, 2005). The evidence suggests that individual health deficits grow exponentially with age in advanced countries (e.g. Mitnitski et al., 2002; Harttgen et al., 2013). Thus, we assume that the change in the deficit index of a member of cohort ν between period t and $t+1$ is increasing in the deficit index accumulated until period t. The accumulation process is slowed down by receiving health input $E_{v,t}$. The health deficit index evolves according to

$$d_{v,t+1} - d_{v,t} = \begin{cases} \varrho d_{v,t} - \kappa E_{v,t} & \text{if } E_{v,t} < \frac{\varrho}{\kappa} d_{v,t}, \\ 0 & \text{otherwise}, \end{cases} \tag{13}$$

$\kappa > 0$, $\varrho > 0$, with initial value $d_{\min} \equiv d_{v,v} > 0$. Parameter ϱ is the growth rate of the health deficit index in absence of health interventions. It can be interpreted as the physiological "force of ageing".[17] κ is a shift parameter we employ to calibrate the model.

We conceptualize health input, $E_{v,t}$, as individual health good consumption to treat illnesses that are caused by existing health deficits, weighted by the quality of the consumed health goods. We thereby capture that health deficits derive from past, not fully cured or not fully curable illnesses. For illustration, consider two health deficits within the set of potential health deficits in the empirical gerontology literature that motivates

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[16]We will calibrate the model such that the initial period for each cohort member is at the age of 20.

[17]Health deficit accumulation would cease if the health input became sufficiently high. Although this may not pure utopia but conceivable with further biotechnological advances (De Grey and Rae, 2007), the case does not arise in our calibrated model.

our modeling approach. First, the physical difficulty to move is known to contribute to developing cardiovascular diseases. If not treated properly, these lead to further health deficits. Second, feeling lonely may cause major depressive disorder. Again, without treatment, further health deficits develop.

Formally, an individual born in v acquires set $\mathcal{I}_{v,t} \subset [0,1]$ of illnesses in period $t \geq v$. Its measure relates to the current deficit index, $|\mathcal{I}_{v,t}| = d_{v,t}$. We normalize the maximally effective individual consumption per health good in a given period to unity. For instance, in the case of pharmaceuticals there is an optimal dose. We capture under-utilization of health care by allowing the actual consumption for any health good to be smaller than unity. The "health care provision wedge" in t is parameterized by $\varphi_t \in [0,1]$. One reason of under-utilization is institutionally caused health care rationing. Full utilization is possible only without rationing. In this case, $\varphi = 0$, whereas $\varphi = 1$ holds in absence of a health system or full exclusion from it. By the law of large numbers, suffering from a set of illnesses $\mathcal{I}_{v,t}$ of measure $d_{v,t}$ in $t \geq v$, an individual born at v thus receives health input

$$E_{v,t} = (1 - \varphi_t) \int_{j \in \mathcal{I}_{v,t}} q_t(j)\mathrm{d}j = (1 - \varphi_t)d_{v,t}Q_t. \tag{14}$$

It depends on the interaction between the contemporaneous health care utilization $(1 - \varphi_t)$, the current deficit state $(d_{v,t})$ and the average quality of health goods (Q_t). Substituting (14) into (13), the growth rate of the health deficit index is deterministic and independent of the deficit state. For $t \geq v$ it is given by

$$\frac{d_{v,t+1} - d_{v,t}}{d_{v,t}} = \begin{cases} \varrho - (1 - \varphi_t)\kappa Q_t & \text{if } Q_t < \frac{\varrho}{\kappa(1-\varphi_t)} \equiv \bar{Q}_t, \\ 0 & \text{otherwise.} \end{cases} \tag{15}$$

Each surviving member of cohort v in period t consumes

$$h_{v,t} = (1 - \varphi_t)S_{v,t}d_{v,t} \tag{16}$$

units per health good from the latest vintages (i.e. an average dose $1 - \varphi_t$ for any member of cohort v and for each illness $j \in \mathcal{I}_{v,t}$). Total demand for each selected vintage of a health good in period t is given by the summing up $h_{v,t}$ over all cohorts with living members:

$$H_t = \sum_{v=t-T+1}^{t} h_{v,t} = (1 - \varphi_t) \sum_{v=t-T+1}^{t} d_{v,t} S_{v,v} \prod_{u=v}^{t-1} (1 - m_{v,u}), \tag{17}$$

where we used (2) and (16) for the latter equation. Thus, more rationing in health care provision (a higher φ) saves health costs by reducing health good consumption, all other things being equal. However, a higher φ has two detrimental effects on health status and life expectancy. First, according to (15), it speeds up the evolution of health deficits for a given stock of medical knowledge, Q. Second, according to (17), it lowers market size for health goods, H, in turn reducing incentives for health innovations.

4 Positive Analysis

We first highlight some equilibrium conditions and then conduct a supply-side analysis of the calibrated model.

4.1 Preliminaries

Denote by π_t the instantaneous profit of health good producers, which are all identical *ex ante* and thus also *ex post*. Ruling out bubbles and arbitrage possibilities in the financial market and accounting for the probability μ_u that health good producers are driven out of business in period $u \geq t + 1$, the value of a vertical innovation in t reads as

$$V_t \equiv \pi_t + \sum_{u=t+1}^{\infty} \frac{\prod_{s=t+1}^{u} (1 - \mu_s) \pi_u}{(1 + \bar{r})^{u-t}}. \tag{18}$$

Labor market clearing implies that

$$L_t^Y + L_t^H + L_t^Q = L_t, \tag{19}$$

where $L_t^H \equiv \chi H_t$ denotes total employment in health goods production. For later use, denote employment shares by $\ell_t^Y \equiv L_t^Y/L_t$, $\ell_t^H \equiv L_t^H/L_t$ and $\ell_t^Q \equiv L_t^Q/L_t$, i.e. in equilibrium, $\ell_t^Y + \ell_t^H + \ell_t^Q = 1$.

As implied by the assumption that the interest rate is exogenous, consumer choices (introduced in section 4) do not play a role for the allocation of labor, health costs, longevity and morbidity. The dynamical system and the long run equilibrium are summarized in Appendix A.

For later use, denoting the gross domestic product (GDP) by $GDP_t \equiv Y_t + p_t H_t$, the health expenditure share reads as

$$\mathfrak{s}_t \equiv \frac{p_t H_t}{GDP_t} = \frac{p_t H_t}{Y_t + p_t H_t}. \tag{20}$$

Also, let us define the "dependency ratio", DPR, as the ratio of retirees to workers. Denoting the size of the retired (old-aged) population by $O_t \equiv \sum_{u=t-T+1}^{t-R} S_{u,t}$, we have

$$DPR_t \equiv \frac{O_t}{L_t} = \frac{\sum_{u=t-T+1}^{t-R} S_{u,t}}{\sum_{u=t-R+1}^{t} S_{u,t}}. \tag{21}$$

4.2 Calibration

We dynamically calibrate the model to endogenous observables in the UK whenever available; otherwise we use North American data. We assume as the baseline calibration that future urbanization and better information about treatment possibilities of patients will continue to lead to slightly improve health care usage over the next decades, despite the counteracting force of increased health care rationing measures. This means that health care wedge φ_t is slightly decreasing in the next decades. The calibration details

are relegated to Appendix B. In the reform scenario, we will assume that future rationing pursues the goal of stabilizes the health expenditure share such that φ_t is increasing in the next decades.

The calibrated model fits UK survival functions quite well, as shown in Figure 1. The most important deviation from the data (solid lines) is for middle-aged individuals in 1950 and to a lesser degree in 1970. Importantly, we use the cross-section of mortality rates for a given year rather than those for a given cohort over time. Doing so is consistent with the standard way of computing "period life expectancy", but different to $S_{v,t}$ in the theoretical model.[18] In contrast to the alternative and theoretically correct concept of "cohort life expectancy", it does not account for changes in access or quality to health care over time, that would alter future mortality rates.

The implied health expenditure share in GDP ($\mathfrak{s}_t$) is 5.0 percent in 1980, 5.1 percent in 1990, 6.2 percent in 2000 and 8.3 percent in 2010, compared to the observed UK levels of 5.1, 5.1, 6.3 and 8.5 percent, respectively (OECD, 2016). The rate of change of the health deficit index across cohorts implied by the calibration is 3.8 percent. According to Mitnitski et al. (2002a), the estimated rate of change of the health deficit index at a given year in the cross-section of Canadian cohorts is equal to 4.3 percent for men and 3.1 percent for women. Finally, we may approximate ℓ_t^H with the employment share in human health activities, as published by the OECD. For the UK, in 2010, it was 7.3 percent.[19] Including additionally residential care and social work activities (that may include other activities than health care provision) would suggest that ℓ^H was 12.7 percent. Our calibrated model gives us a value in-between, equal to 10 percent in 2010.

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[18]Corresponding to Figure 1, Table A.2 in the Online-Appendix compares in detail the remaining "period life expectancy" at a given age implied by the calibrated model with the empirical ones in the UK.

[19]See http://stats.oecd.org/Index.aspx?DataSetCode=SNA_TABLE3, retrieved on January 31, 2016.
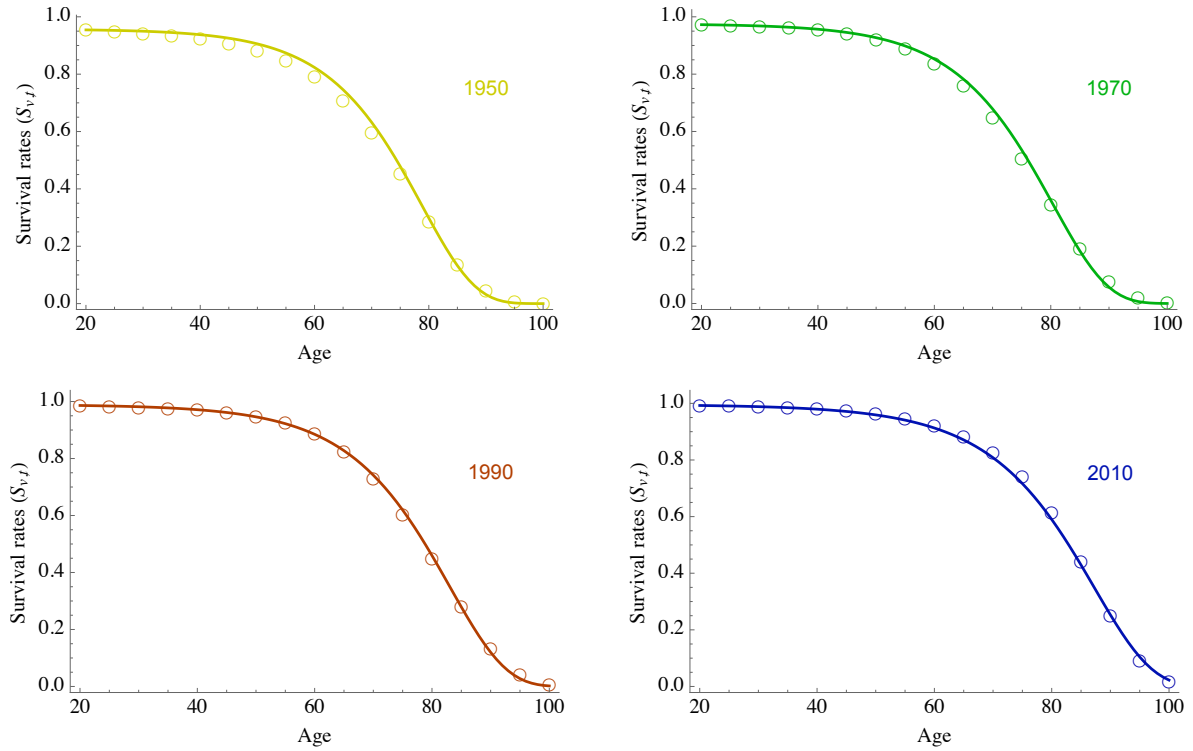
Figure 1: Survival curves for 1950, 1970, 1990 and 2010: Calibrated model vs. UK data.

Notes: (1) Empirical series: solid lines, calibrated model: circles (2) Data source: www.mortality.org. (3) Time paths $\{\varphi_t\}$ and $\{S_{v,v}\}$ are displayed in Figure A.2 (Online-Appendix). (4) Initial quality index (in 1870) $Q_0 = 0.01 \cdot \lim_{t\to\infty} Q_t$ for $\lim_{t\to\infty} \varphi_t = 0.05$. (5) Other parameters: $\alpha = 0.38$, $\delta^K = 0.07$, $\sigma = 1.5$, $\phi = 2.65$, $\chi = 0.9$, $\varrho = 0.04$, $\kappa = 0.06$, $\xi = 0.065$, $\eta = 0.12$, $\delta^Q = 0.02$, $\vartheta = 0.6$, $g = 0.02$, $\bar{r} = 0.05$, $d_{\min} = 0.03$, $d_{\max} = 0.67$, $\gamma = 0.1$, $\Gamma = 1.25$, $T = 101$, $R = 43$.

4.3   Results

We now examine for alternative policy scenarios the future evolution (for periods $t \geq t_0$) of age-specific survival rates (based on the "period mortality rates" across cohorts for a given year), age-specific morbidity ($d_{v,t}$), age-specific health care demand ($h_{v,t}$), the total health expenditure share ($\mathfrak{s}_t$), the employment structure (ℓ_t^H, ℓ_t^Q), and the old-age dependency ratio (DPR_t). We also investigate how age-specific (period and cohort) life expectancies change over time.

4.3.1 Baseline Scenario

We start with the implications of the baseline scenario for the future. Panel (a) of Figure 2 displays implied survival curves for 2020 (solid black line), 2050 (dashed blue line) and 2080 (dotted green line), suggesting that they are considerably shifting upwards over time. Rising survival rates are driven by declining morbidity, displayed in panel (b). This is because age-specific mortality rates $(m_{v,t})$ decrease when health deficits $(d_{v,t})$ accumulate with age at lower rates, according to (1). For instance, the health deficit index (the fraction of actual health deficits out of a set of possible deficits) for someone having reached age 80 declines from 18.5 percent in 2020 to 12.9 percent in 2050 and 9.7 percent in 2080. According to (15), the ageing process is slowed down if the stock of medical knowledge (Q_t) is endogenously increasing or if there is better access to health care. Both forces are present in the baseline scenario.

The evolution of health deficits $(d_{v,t})$ determines, in interaction with survival rates $(S_{v,t})$, the evolution of age-specific health care demand $(h_{v,t})$, according to (16). As displayed in panel (c), total age-specific health care demand is inverted U-shaped as a function of age.[20] Over time it shifts to the right. That is, health care demand decreases for younger individuals and increases for older ones. The shift reflects that, for younger individuals, improvements in the quality of health goods have little effect on survival rates, whereas the opposite holds for older individuals. In fact, survival rates of younger individuals are high and their deficit index is low to begin with. By contrast, total health care demand for older age-groups is rising over time because of considerable increases in survival rates.

Consequently, despite declining morbidity and declining mortality at any age, population ageing may result in increasing health expenditure shares $(\mathfrak{s}_t)$ over time. According to panel (d), the health expenditure share increases from 8.4 percent in 2020 to 9.2 percent in 2050 and 10.3 percent in 2080. Panel (e) shows that this is associated with a

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[20]For panel (c) of Figure 1, we use the true "cohort mortality rates" $(m_{v,t})$ for computing survival rates $(S_{v,t})$ rather than "period mortality rates", unlike for panel (a).

comparable increase in the health employment share (ℓ_t^H). Importantly, it also raises incentives for health innovations through increased market size. This implies that the medical R&D labor share (ℓ_t^Q) is rising over time as well, according to panel (f). According to (11), this leads to improvements in the quality of health care (Q) that is strongly associated with the displayed evolution of morbidity and mortality.

As is well known and reflected in (21), demographic change induced by human ageing leads to a rising old-age dependency ratio (DPR_t) over time. The interesting question is by how much. Projections in the literature that do not account for the endogeneity of health care quality and possible changes to health care access are not very informative in this respect. Panel (i) shows the evolution of the ratio of population size aged 63+ (retirement age) to the population size aged 20-62 (working age). It suggests that DPR rises from 45 percent in 2020 to 65.2 percent in 2050 and 88.9 percent in 2080. Thus, our model implies that the ratio of retirees to workers will be doubling in the next 60 years.

In sum, our model gives rise to an important insight that has yet not been clearly worked out in the literature: population ageing that is associated with health improvements at any age may be associated with rising health expenditure shares even if prices of health goods grow at the same rate than income.[21] In this sense, rising health costs are good news. As we will argue in the next section (welfare analysis), therefore, measures to raise health care rationing may not be desirable.

4.3.2 Reform Scenario: Stable Health Expenditure Share

Before doing so, we analyze the consequences of a health care rationing scheme that stabilizes the health expenditure share from year 2020 onwards (i.e. $\mathfrak{s}_t = \mathfrak{s}_{t_0}$ for $t \geq t_0$). It requires a substantial increase in the health care provision wedge (φ_t) over time, from 11 percent in 2020 to 17 percent in year 2050 and 27.2 percent in year 2080.[22] The implications can be seen in Figure 3.

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[21]Recall that labor income is the exclusive source of health care finance in our model and health good prices grow at the same rate than wage rates by construction.

[22]The scheme is displayed in Figure A.3 of the Online-Appendix.
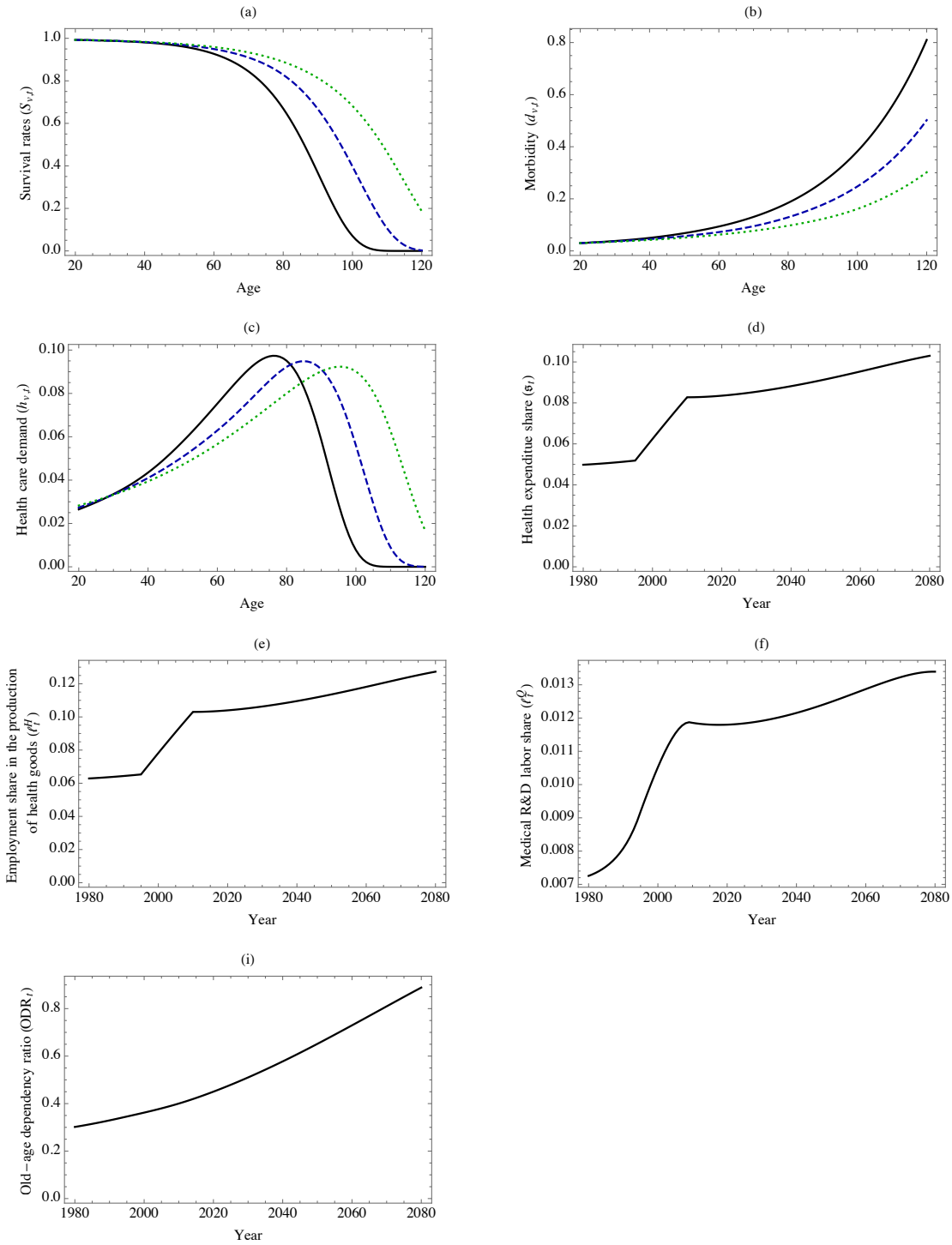
Figure 2: The future of human health, longevity and health costs for the baseline policy scenario.

Notes: (1) Panels (a)-(c): Solid (black) line for 2020, dashed (blue) line for 2050, dotted (green) line for 2080. (2) Parameters as for Figure 1.

The thin lines in panels (a)-(c) repeat the results for the baseline scenario shown in Figure 2, whereas the thick lines correspond to the reform scenario with extended health care rationing. Panel (a) suggests that survival rates improve to a lesser degree than in the baseline scenario. The differences across policy regimes are particularly visible for 2080, whereas differences are small for 2050. The same is true with respect to morbidity $(d_{v,t})$, according to panel (b). Panel (c) shows that age-specific health care demand $(h_{v,t})$ is reduced compared to the baseline scenario, particularly for older age-groups. This reflects that survival rates of older cohorts improve less over time which dominates the effect that morbidity is higher for any age.

In panels (d)-(g), the thin lines again repeat the results for the baseline scenario shown in Figure 2, whereas the thicker, dashed lines correspond to the reform scenario. Panel (d) displays the, by design, time-invariant health expenditure share $(\mathfrak{s}_t)$ in the reform scenario. According to panel (e), consequently, also the employment share in the production of health goods (ℓ_t^H) is basically time-invariant. Panel (f) shows that the medical R&D labor share (ℓ_t^Q) even decreases slightly over time. That is, compared to the baseline scenario, medical R&D effort is considerably reduced. Such dynamic incentive effect of health care rationing adds to the static reduction in health care usage to jointly slow down both demographic change and health improvements in the population. According to panel (g), consequently, the old-age dependency ratio (DPR_t) is rising more moderately than in the baseline scenario, from 45 percent in 2020 to 64.6 percent in 2050 and 83.6 percent in 2080.

4.3.3 Comparing Life Expectancy Effects

We can compare age-specific (remaining) life expectancies from the age-specific mortality rates for the two scenarios in two ways. First, we can calculate for both scenarios the "period life expectancy", as it is usually done in the literature (e.g. Kontis et al., 2017). For this, like for the displayed survival rates, we use the "period mortality rates" from the cross-section of cohorts and pretend they stay constant over time. As will become
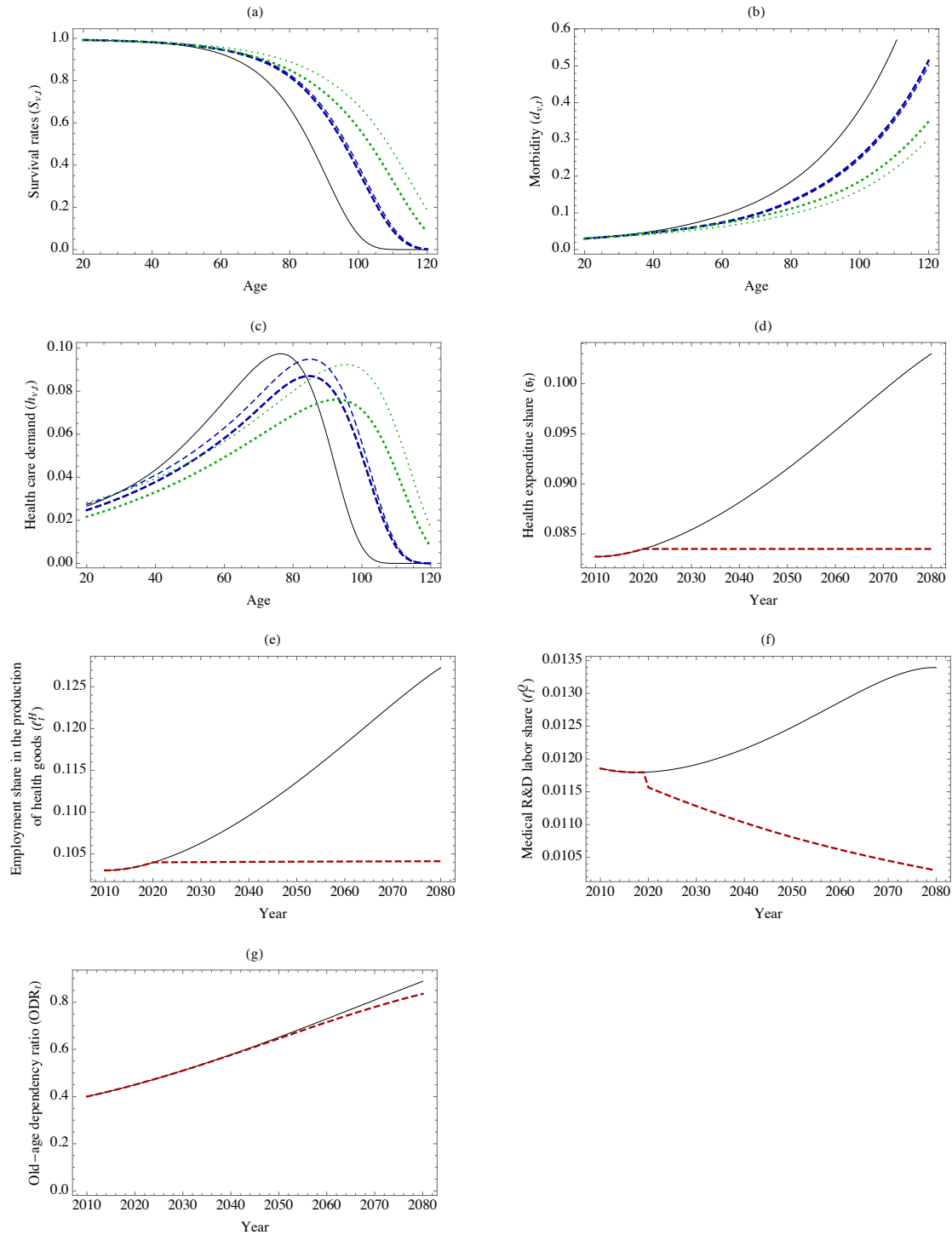
Figure 3: Effects of extending health care rationing from year 2020 onwards for stabilizing the health expenditure share (reform scenario).

Notes: (1) Panels (a)-(c): Solid (black) line for 2020, dashed (blue) line for 2050, dotted (green) line for 2080. Thin lines repeat the baseline scenario, thick lines show the reform scenario. (2) Panels (d)-(g): Thin (black) lines repeat the baseline scenario, dashed (red) lines show the reform scenario. (3) Time paths for $\{\varphi_t\}$ in the two scenarios as displayed in Figure A.3 (Online-Appendix). (4) Other parameters as for Figure 1.

24

apparent shortly, this dramatically underestimates life expectancy when access or quality to health care significantly improves over time. Second, therefore, what we are really interested in is "cohort life expectancy", based on the correct age-specific mortality rates $(m_{v,t})$ in the future.

Remaining cohort life expectancy of a member of a cohort born in v from t onwards is computed as follows. Recall that the number of persons surviving to age $t-v$ is $S_{v,t}$ as given by (2). We calculate the "person-years lived" between ages $t-v$ and $t-v+1$ when born in v as $\mathcal{P}_{v,t} \equiv S_{v,t+1} + 0.5 \cdot S_{v,t} m_{v,t}$, where $S_{v,t} m_{v,t}$ is the number of persons dying between age $t-v$ and $t-v+1$ from the cohort born in v. Then $\mathcal{N}_{v,t} \equiv \sum_{u=t}^{v+T-1} \mathcal{P}_{v,u}$ is the total number of years lived after attaining age $t-v$. Remaining life expectancy at age $t-v$ is given by $\mathcal{N}_{v,t}/S_{v,t}$.

Period Life Expectancy Figure 4 displays life expectancy in a given yeaar for someone having reached age 20 and someone aged 65 in both the baseline scenario (solid line) and the reform scenario (dashed line). The circles show the evolution of the respective empirical period life expectancies in the UK, underlying how well the calibrated model fits the data.

For the baseline scenario, we find that an individual that has reached age 20 in year 2020 (i.e. was born in 2000) can expect to live until age 83.6 under the (invalid) assumption that age-specific mortality rates in a given year will not improve over time. Analogous figures are 93.5 and 103.7 years when reaching age 20 in 2050 and 2080, respectively.[23] Moreover, someone having reached age 65 in 2020, 2050 and 2080 can expect to live until age 86.9, 96.2 and 106.2, respectively.

With the considered health care reform that stabilizes the health expenditure share, period life expectancy increases less than in the baseline scenario. The difference across scenarios is 0.8 years and 4.6 years for someone reaching age 20 in year 2050 and 2080, respectively, and 0.7 and 4.0 years for someone having reached age 65 in 2050 and 2080,

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[23]See Table A.3 in Online-Appendix (left columns) for the remaining age-specific "period life expectancies".

respectively.[24]

In sum, the model suggests for both scenarios considerable gains in period life expectancy over time. Shorter run effects from implementing the cost-saving health care reform are smaller than longer run effects. Over time, the reform induces sizable reductions in life expectancy gains.
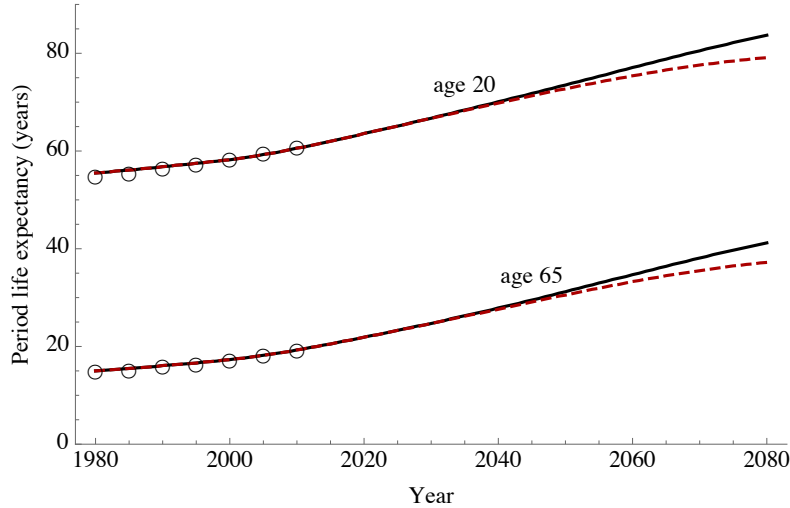

Figure 4: Implied remaining period life expectancies at age 20 and age 65: baseline vs. reform scenario.

Notes: (1) Solid (black) line for baseline, dashed (red) line for reform, circles according to UK data. (2) Parameters as for Figure 2 (baseline) and Figure 3 (reform).

Cohort Life Expectancy Figure 5 examines the cohort life expectancies at age 20 and 65, i.e. fully taking into account changes in age-specific mortality rates over time. First, we see that in both scenarios the (conceptually correct) life expectancy for those cohorts born already are considerably higher than period life expectancy. Someone with age 20 in year 1980 (i.e. born in 1960) can expect to live until age 91.1 years in the baseline scenario and 90.3 years in the reform scenario when taking into account favorable future reductions in mortality rates. The static view displayed in Figure 4 would underestimate the remaining life expectancy in both scenarios by more than 14 years. In the baseline scenario, someone who is 20 years old in 2020 can expect to die at age 106.2 whereas

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[24]Again, see Table A.2 in Online-Appendix (right columns).

period life expectancy is 22.6 years shorter. Someone with age 65 in 1980 could have expected to live 16.4 additional years in both scenarios, whereas according to period life expectancy it was 15 years. Clearly, the error by taking period life expectancy rather than cohort life expectancy is much smaller for higher ages. It reflects that the time frame to profit from improvements in both the quality of health goods and access to health care is smaller, given that an individual accumulates health deficits grow exponentially when holding Q and φ constant.

Second, the difference in the evolution of life expectancy across scenarios is considerably higher in Figure 5 compared to Figure 4. An individual that has reached age 20 in year 2050 can expect to live until age 111 in the baseline scenario and until age 100.6 in the reform scenario, i.e. one decade less.[25] Thus, the concept of period life expectancy severely underestimates potential life expectancy effects of health care rationing (recall that the difference in period life expectancy across scenarios in the same case is only 0.8 years). Someone who is 65 in 2050 can expect to live until age 106.1 in the baseline scenario and 3.7 less in the reform scenario. Hence, like for period life expectancy, the loss in remaining life expectancy from stabilizing the health expenditure share is lower for older persons.

5   Normative Analysis

In this section, we examine the welfare implications of the switch in health policy from the baseline scenario (analyzed in Figure 2) to the reform scenario with stabilized health expenditure share from 2020 onwards (Figure 3). For concreteness, we assume that the policy regime switch is not anticipated by living members of generations born before the shock.

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[25]See Table A.4 in Online-Appendix. Our predictions may be compared to the estimates of cohort life expectancy by the Office for National Statistics (2015) for the UK. It suggests that a female who has reached age 20 in year 2050 can expect to live until age 109.1 in the most optimistic of three scenarios (2.1 years longer than a comparable male), but only until age 85.5 years in the most pessimistic scenario (2.9 years longer than a comparable male).
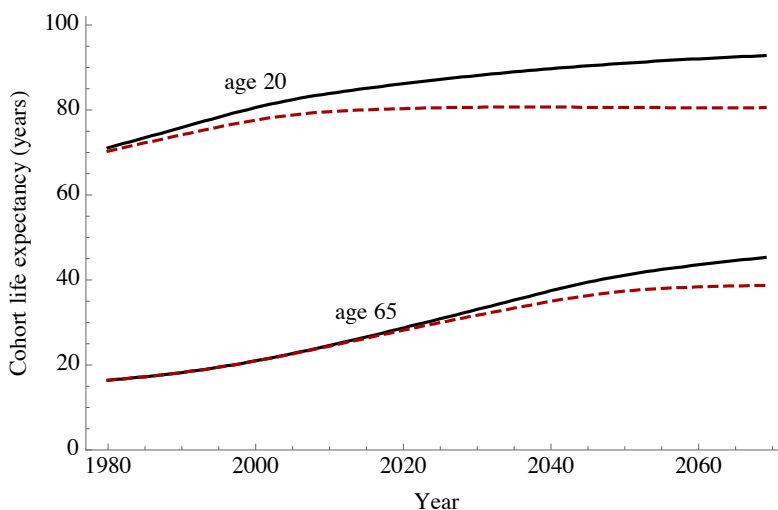
Figure 5: Implied remaining cohort life expectancies at age 20 and age 65: baseline vs. reform scenario.

Notes: (1) Solid (black) line for baseline, dashed (red) line for reform. (2) Parameters as for Figure 2 (baseline) and Figure 3 (reform).

5.1 Welfare Behind the Veil of Ignorance

We first need to define an appropriate welfare criterion. We consider welfare behind the veil of ignorance. Facing uncertain death, rational individuals calculate (under rational expectations) the expected utility from life-time consumption by multiplying the instantaneous utility u experienced in a given period with the probability to survive beyond that period ($S_{v,t}$). Instantaneous utility, u, positively depends on consumption level of the numeraire and negatively depends on the health deficit index.

Formally, with maximum life span T, a member of cohort v has preferences that are represented by the intertemporal utility function

$$U_v = \sum_{t=v}^{v+T-1} \beta^{t-v} S_{v,t} u(c_{v,t}, d_{v,t}), \tag{22}$$

where $c_{v,t}$ denotes the consumption level. Instantaneous utility is given by

$$u(c_{v,t}, d_{v,t}) \equiv \frac{\log c_{v,t}}{(1 + d_{v,t})^\zeta}, \qquad (23)$$

where $\beta \geq 0$ is the discount factor and $\zeta > 0$ measures to which extent a higher deficit state reduces the marginal utility of consumption. For an individual without health deficits ($d_{v,t} = 0$) or in the case where $\zeta = 0$, we are back to a standard instantaneous utility function. With log-utility, the intertemporal elasticity of substitution is unity, as supported by Chetty (2006), among others.

We assume that the health care system is financed by a constant contribution rate out of wage income, denoted by τ_t for period t.[26] The health care budget is balanced in each point in time; that is, revenue, $\tau_t w_t L_t$, equals expenses, $p_t H_t$. Consequently, recalling (12), the health contribution rate equals the mark-up factor for health goods (Γ) times the share of labor (ℓ^H) allocated for producing health goods and services:

$$\tau_t = \Gamma \ell_t^H. \qquad (24)$$

Denote asset holding ("wealth") of a member of cohort v in t by $a_{v,t}$. Initial asset holding is $a_{v,v} = 0$ since there is no bequest motive and the annuity market is perfect. We assume fair insurance within a cohort in the annuity market. That is, zero-profit insurance companies pay a rate of return above $\bar{r}$ but keep the individuals' wealth after death. The corresponding law of motion for an individual of cohort v, wealth at $t \geq v$ can be written as

$$a_{v,t+1} = (1 - \tau_t)w_t + (1 + r_{v,t})a_{v,t} - c_{v,t}, \qquad (25)$$

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[26] Assuming that health insurance is paid by workers and enjoyed by retirees greatly simplifies the analysis. If health insurance were also be financed by capital income, we would have to keep track of aggregate asset holdings in the economy. Recall that these are unrelated to investments as the interest rate is fixed at $\bar{r}$.

where the cohort-specific interest factor between date t and $t+1$ is given by[27]

$$1 + r_{v,t} = \frac{1 + \bar{r}}{1 - m_{v,t-1}}. \tag{26}$$

Individuals of each generation v choose their consumption paths $\{c_{v,t}\}_{t \geq v}$ to maximize utility U_v s.t. (25) and non-negativity constraint $a_{v,v+T} \geq 0$. They have perfect foresight about the health contribution rate and health deficit states (including implied mortality risks in (1)) that result in the baseline policy regime and take these as given when optimizing.

The policy reform of extended rationing, assumed to apply in period t_0 (again, in year 2020) is assumed to be unanticipated. That is, living members of generations $v < t_0$ (i.e. those already born) re-optimize. All agents have perfect foresight of the new policy regime from t_0 onwards. The optimization problems of both generations $v < t_0$ and $v \geq t_0$ are solved in Appendix C.

Welfare effects of policy reforms are evaluated as follows. Let superscript 0 on consumption levels, deficit states and survival rates denote the values of these variables in the baseline policy regime and superscript 1 the values in the policy reform regime. Moreover, let

$$U_v^i(\psi) \equiv \sum_{t=v}^{v+T-1} \beta^{t-v} S_{v,t}^i \frac{\log(\psi c_{v,t}^i)}{\left(1 + d_{v,t}^i\right)^\zeta} \tag{27}$$

denote the life-time utility of cohort v when consumption levels in scenario $i \in \{0, 1\}$ are multiplied with factor $\psi > 0$. By definition (27), life-time utility in the reform scenario is $U_v^1(1)$. We report cohort-specific factors ψ_v that solve

$$U_v^0(\psi_v) = U_v^1(1). \tag{28}$$

Thus, ψ_v is the equivalent variation (EV) welfare measure in the baseline scenario such that cohort v gets the same utility as in the reform scenario.[28]

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[27]See e.g. Heijdra, Mierau and Trimborn (2016).

[28]See Jones and Klenow (2016) for a similar way to measure welfare differences of randomly chosen

5.2 Calibration

We choose a typical value for the subjective discount rate, β, such that $\beta(1+\bar{r}) > 1$,[29] setting $\beta = 0.98$ (recall $\bar{r} = 0.05$). Next, we calibrate ζ, which determines the loss in marginal utility from consumption caused by health deficits. Finkelstein et al. (2013) find that, starting at the mean, a one-standard deviation increase of chronic diseases is associated with a decline in the marginal utility of consumption, denoted by $LOSS$, of 11.2 percent. Marginal consumption utility reads as $(1+d_{v,t})^{-\zeta}/c_{v,t}$. Evaluated at the mean deficit index, $\mathbb{E}(d)$, and denoting the standard deviation by $\mathcal{STD}(d)$, the estimate of Finkelstein et al. (2013) then suggests that ζ is given by

$$\frac{[1 + \mathbb{E}(d) + \mathcal{STD}(d)]^{-\zeta}}{[1 + \mathbb{E}(d)]^{-\zeta}} = 1 - LOSS. \tag{29}$$

According to Mitnitski et al. (2002), the mean deficit index in the population is $\mathbb{E}(d) = 0.054$ and the standard deviation is $\mathcal{STD}(d) = 0.024$. Hence, $\zeta = -44.42 \cdot \log(1-0.112) = 5.1$.

Finally, we have to calibrate the initial general state of technology, A_0. This is because the time path of productivity and wage income potentially affects welfare changes from the policy reform. We do this by targeting a certain ratio of the value of life to GDP per worker. Denote the value of life of an individual born in v by W_v and assume it is given by expected (indirect) life-time utility in the baseline scenario for a cohort with age 20 in 2010 normalized by the marginal instantaneous (indirect) utility in the initial period of life:

$$W_v \equiv \frac{U_v^0(1)}{\frac{\partial u(c_{v,v}, d_{\min})}{\partial c}} = U_v^0(1)(1 + d_{\min})^\zeta c_{v,v}. \tag{30}$$

Assuming that A_t grows annually at rate $g = 0.02$, we set A_0 such that, for the year 2010, W_v/y_v equals 60, 80 or 100.[30] Given that GDP per person employed in the UK

individuals in a cross-country context rather than across policy regimes.

[29]If we assumed $\beta(1 + \bar{r}) = 1$, then individual consumption would monotonically decrease with age, which is inconsistent with the evidence.

[30]The value of life is an inherently normative concept. Any attempt to compute it in the literature

was about 75,000 US$ (PPP) in 2010, these ratios correspond to a value of life of 4.5, 6 and 7.5 million US$, respectively.

5.3   Results

Figure 6 displays the cohort-specific welfare effects (EV) of switching from the the baseline scenario (analyzed in Figure 2) to the reform scenario (Figure 3). For older cohorts (born in the 1960s), the policy reform is almost neutral for welfare. On the one hand, those close to retirement age at the time of the reform do not save much health care contributions (that we assumed to be entirely paid by workers) from the reform. On the other hand, the detrimental effects from the reform on longevity and morbidity are small for individuals who have accumulated a lot of health deficits already.

For later cohorts, however, the welfare change can becomes substantially negative. This is remarkable since younger cohorts save health contributions over a long working period. Those who start working life after the reform year 2020 benefit from reduced contributions even for the entire working life, whereas reductions in survival rates in response to the reform are minor for working-aged individuals. However, reduced survival rates during retirement and reduced instantaneous utility from higher health deficits by far outweigh the utility increases from higher disposable income for younger generations. We estimate that someone who is 20 years old in 2020 would experience a reform-induced welfare loss of 20.4 percent when assuming the medium ratio of the value of life to GDP per worker. Assuming alternatively a lower or higher ratio, the losses are 16.1 and 24.4 percent, respectively. The losses are even higher for future generations. Someone who is 20 years old in 2050 would experience a corresponding welfare loss of in the range of 34.4-47.8 percent (41.5 percent in the medium case). Such dramatic welfare loss from extending health care rationing reflects the sizable losses in cohort life expectancy from

---

has necessarily been based on strong assumptions. For instance, Hall and Jones (2007) assume that the value of an additional year lived equals the health costs to increase life expectancy by an additional year. This implicitly assumes that the US health system is optimal in the sense of equating marginal benefits and marginal costs of saving lifes.

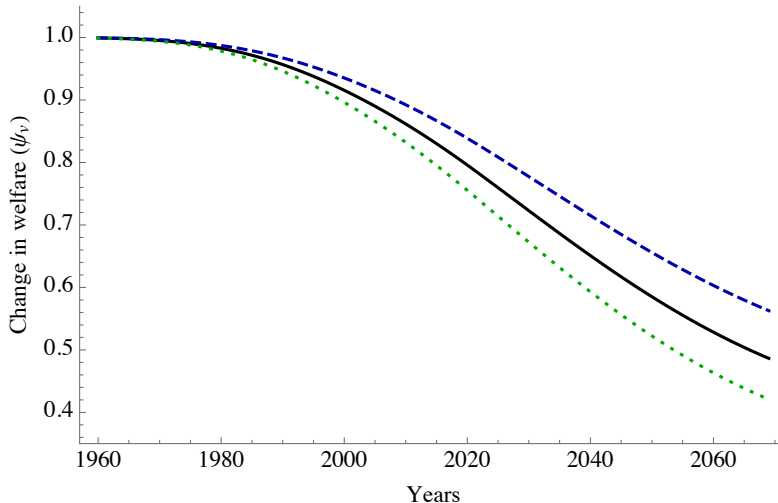the reform (displayed in Figure 5) as well as increased morbidity (displayed in panel (c) of Figure 3).

Figure 6: Cohort-specific welfare effects (EV) of extending health care rationing for stabilizing the health expenditure share in three alternative cases.

Notes: (1) For instance, the displayed change in welfare evaluated at year 2020 corresponds to the EV of the cost-saving reform for someone who is 20 years old in 2020. (2) Solid (black) line: medium ratio of value of life to GDP per worker, dashed (blue) line: low ratio, dotted (green) line: high ratio, (3) $\beta = 0.98$, $\zeta = 5.1$. $A_0 \in \{1.55, 6, 23\}$ for small, medium, high ratio of value of life to GDP per worker. (4) Time paths $\{\varphi_t\}$ in baseline scenario 0 and reform scenario 1 are displayed in Figure A.3 (Online-Appendix). (5) Other parameters as for Figure 1.

6 Concluding Remarks

We studied the interdependence of medical R&D, health expenditure, longevity and morbidity of an age-structured population in a novel, multi-period overlapping generations model. Without health care reform, our calibrated model suggests a rising health expenditure share in GDP at the benefit of both substantial future increases in human longevity and reductions in morbidity.

The key to perform such analysis is to capture biologically founded ageing, based on gerontology research. Our approach has two advantages. First, it enables us to calibrate the model by using the health deficit index as simple and observable measure of health

status. It has proven being a powerful correlate of mortality rates. Second, the approach captures the empirically established path-dependency of health deficits. It implies that improperly treated health deficits lead to new ones that, overall, may considerably shorten life-time.

The path-dependency of health deficits has important consequences for the desirability of extending health care finance that results from medical advances and associated gains on longevity. The standard reasoning in the debate on health care rationing was that some treatments like hip replacements are only affecting quality of life but not life expectancy and thus would be expendable. Such view has proven utterly wrong by gerontology research. For instance, the physical difficulty to move is known to contribute to developing cardiovascular diseases that shortens life expectancy.

Our analysis suggests that stabilizing the health expenditure share by extending health care rationing has potentially sizable effects on morbidity and longevity particularly in the longer run. The implied welfare effects of extending health care rationing may be substantially negative for future generations. Whereas shorter run effects can mainly be attributed to the direct effects of health care rationing on the accumulation of health deficits, longer run implications also work through reduced medical R&D incentives. That is, population ageing and rising health costs interact with each other through the market size effect of increased life expectancy on medical technological progress. Thus, rising health expenditure shares are not a problem but a blessing for human health and longevity that is fueled by medical R&D.

In future research we aim to allow for the possibility of private purchases of health goods and services in a health care system with rationing. Its consideration would naturally refocus the debate on health inequality issues, for instance, when purchases of life-saving drugs may be available only for richer individuals. Such policy regime could give rise to major distributional conflicts. The associated challenges for modern societies appear profound and discomforting.

Appendix

A. Dynamical System and Long Run Equilibrium

- **Dynamical System:** Recall that $V_{t+1}(j)$ is the value of an innovation in health sector j resulting from R&D effort in t. A representative R&D firm searching for a vertical innovation to treat illness j solves

$$\max_{l_t(j)}\{\mu_{t+1}(j)V_{t+1}(j) - w_t l_t(j)\} = \left(\tilde{\xi}_t V_{t+1}(j) - w_t\right) l_t(j), \tag{31}$$

according to (7). Thus, $\tilde{\xi}_t V_{t+1}(j) = w_t$ for all j. Thus, in equilibrium, R&D firms do not earn profits. Moreover, $l_t(j) = L_t^Q$ and $V_{t+1}(j) = V_{t+1}$ are the same for all $j \in [0,1]$. Using $\tilde{\xi}_t = \xi \cdot (L_t^Q)^{-\vartheta}$, the zero-profit condition for R&D firms reads as

$$V_{t+1}\xi(L_t^Q)^{-\vartheta} = w_t. \tag{32}$$

Given that there is a unit mass of health sectors, the total and per firm amount of labor allocated to the production of health goods is given by $L_t^H = \chi H_t$. Thus, with mark up Γ, the profit per health good producer is

$$\pi_t = (p_t - \chi w_t)\, H_t = (\Gamma - 1)\chi w_t H_t = (\Gamma - 1)w_t L_t^H, \tag{33}$$

according to (12). According to (18),

$$\begin{aligned} V_t &= \pi_t + \frac{1 - \mu_{t+1}}{1 + \bar{r}}\pi_{t+1} + \frac{(1 - \mu_{t+1})(1 - \mu_{t+2})}{(1 + \bar{r})^2}\pi_{t+2} + \\ &\quad \frac{(1 - \mu_{t+1})(1 - \mu_{t+2})(1 - \mu_{t+3})}{(1 + \bar{r})^3}\pi_{t+3} + ..., \end{aligned} \tag{34}$$

$$V_{t+1} = \pi_{t+1} + \frac{1 - \mu_{t+2}}{1 + \bar{r}}\pi_{t+2} + \frac{(1 - \mu_{t+2})(1 - \mu_{t+3})}{(1 + \bar{r})^2}\pi_{t+3} + ... = \frac{1 + \bar{r}}{1 - \mu_{t+1}}\left(V_t - \pi_t\right). \tag{35}$$

Using (33) in (35), we get the following no-arbitrage condition in the market that

finances health R&D:

$$\frac{1 - \mu_{t+1}}{1 + \bar{r}} \frac{V_{t+1}}{V_t} + \frac{(\Gamma - 1)w_t L_t^H}{V_t} = 1. \tag{36}$$

Now let us define $\mathcal{V}_t \equiv V_t/A_t$. Denote by $\mathfrak{d}_{\mathfrak{a},t}$ the health deficit index of a surviving individual of age $\mathfrak{a}$ in period t and $\widetilde{\mathfrak{a}}_t$ as the highest age in period t such that $\mathfrak{d}_{\mathfrak{a},t} \leq d_{\max}$. Thus, $\bar{\mathfrak{a}}_t \equiv \min(\widetilde{\mathfrak{a}}_t, T)$ is the age at which an individual dies for sure. Neglecting the household side (which is relevant for the welfare analysis only), the dynamical system can be summarized as follows:

$$\mathfrak{d}_{1,t+1} = [1 + \varrho - (1 - \varphi)\kappa Q_t]\, d_{\min}, \tag{37}$$

$$\mathfrak{d}_{2,t+1} = [1 + \varrho - (1 - \varphi)\kappa Q_t]\, \mathfrak{d}_{1,t}, \tag{38}$$

$$\mathfrak{d}_{3,t+1} = [1 + \varrho - (1 - \varphi)\kappa Q_t]\, \mathfrak{d}_{2,t}, \tag{39}$$

$$\vdots$$

$$\mu_{t+1} = \eta Q_t + (1 - \eta Q_t) \cdot \xi \cdot (L_t^Q)^{1-\vartheta}, \tag{40}$$

$$Q_{t+1} - Q_t = \gamma \left(1 - \eta Q_t\right) \xi (L_t^Q)^{1-\vartheta} - (\delta^Q - \gamma\eta)Q_t, \tag{41}$$

$$\frac{1 - \mu_{t+1}}{1 + \bar{r}} \mathcal{V}_{t+1} \left(1 + g\right) + (\Gamma - 1)w_t L_t^H = \mathcal{V}_t, \tag{42}$$

$$\mathcal{V}_{t+1} \left(1 + g\right) \xi \cdot (L_t^Q)^{-\vartheta} = \omega, \tag{43}$$

$$
\begin{aligned}
H_t \;=\; & (1 - \varphi_t)S_{t,t}d_{\min} + (1 - \varphi_t)\left(1 - \tilde{m}(d_{\min})\right) \times \\
& \left\{ S_{t-1,t-1}\mathfrak{d}_{1,t} + \mathfrak{d}_{2,t}S_{t-2,t-2}\left(1 - \tilde{m}(\mathfrak{d}_{1,t-1})\right) + \right. \\
& \mathfrak{d}_{3,t}S_{t-3,t-3}\left(1 - \tilde{m}(\mathfrak{d}_{2,t-1})\right)\left(1 - \tilde{m}(\mathfrak{d}_{1,t-2})\right) + \dots + \\
& \left. \mathfrak{d}_{\bar{\mathfrak{a}}_t,t}S_{t-\bar{\mathfrak{a}}_t,t-\bar{\mathfrak{a}}_t}\left(1 - \tilde{m}(\mathfrak{d}_{\bar{\mathfrak{a}}_t-1,t-1})\right)\left(1 - \tilde{m}(\mathfrak{d}_{\bar{\mathfrak{a}}_t-2,t-2})\right) \times \dots \times \left(1 - \tilde{m}(\mathfrak{d}_{1,t-\bar{\mathfrak{a}}_t+1})\right)\right\}
\end{aligned} \tag{44}
$$

$$L_t^Y + L_t^H + L_t^Q = L_t, \tag{45}$$

according to (15), (9), (11), (36), (32), (17) and (19), respectively, for a given $Q_0 > 0$ and a given vector of current deficit states of the cohorts living in period 0, $\boldsymbol{d}_0 \equiv (\eth_{1,0}, \eth_{2,0}, \eth_{3,0}, ..., \eth_{\bar{a}_0,0})$.

- **Long Run Equilibrium:** We next derive the long run equilibrium (focussing on the case where $Q_{t+1} = Q_t$ holds for $t \to \infty$ only). Setting $Q_{t+1} = Q_t = Q$ in (10) and omitting the time index, we obtain

$$L^Q = \left(\frac{\frac{\delta^Q}{\gamma} Q - \bar{\mu}}{(1 - \bar{\mu})\xi} \right)^{\frac{1}{1-\vartheta}}. \tag{46}$$

Using $\mathcal{V}_{t+1} = \mathcal{V}_t = \mathcal{V}$ in (42) implies

$$\mathcal{V} = \frac{(\Gamma - 1)(1 + \bar{r})\omega L_t^H}{\bar{r} - g + \mu(1 + g)}. \tag{47}$$

Moreover, according to (43) and (46),

$$\mathcal{V} = \frac{\omega(L^Q)^\vartheta}{(1 + g)\xi}. \tag{48}$$

Combining (47) and (48) implies

$$\frac{(L^Q)^\vartheta}{\xi} = \frac{(\Gamma - 1)(1 + \bar{r})L^H}{\frac{\bar{r}-g}{1+g} + \mu} \tag{49}$$

Let $\hat{\eth}_{\mathfrak{a}}$ denote the long run health deficit index of someone of age $\mathfrak{a} \geq 0$, associated with the steady state quality index $\hat{Q} < \bar{Q}$. Moreover, let $\hat{\varphi} \equiv \lim_{t \to \infty} \varphi_t$ and suppose that $\hat{S} \equiv \lim_{v \to \infty} S_{v,v} = 1$. According to (15),

$$\hat{\eth}_{\mathfrak{a}+1} = \left[1 + \varrho - (1 - \hat{\varphi})\hat{Q} \right] \hat{\eth}_{\mathfrak{a}}, \tag{50}$$

37

with initial condition $\hat{\mathfrak{d}}_0 = d_{\min} > 0$. The solution of difference equation (50) gives us the steady state age-path of the health deficit index conditional on φ and $\hat{Q}$, denoted by $\mathfrak{D}(\mathfrak{a}, \varphi, \hat{Q})$, $\mathfrak{a} \geq 0$. Function $\mathfrak{D}(\mathfrak{a}, \varphi, Q)$ is increasing in age, $\mathfrak{a}$, increasing in φ, and decreasing in quality index, Q. Let $\tilde{\mathfrak{a}}_\infty$ denote the largest age $\mathfrak{a}$ such that $\mathfrak{D}(\mathfrak{a}, \varphi, Q) \leq d_{\max}$ and define function

$$\tilde{H}(\varphi, Q) \equiv (1 - \varphi) \left[\sum_{\mathfrak{a}=0}^{\bar{\mathfrak{a}}_\infty} \mathfrak{D}(\mathfrak{a}, \varphi, Q) \prod_{u=0}^{\mathfrak{a}} [1 - \tilde{m}(\mathfrak{D}(u, \varphi, Q))] \right], \qquad (51)$$

where $\bar{\mathfrak{a}}_\infty \equiv \min(\tilde{\mathfrak{a}}_\infty, T)$. Substituting (46) into (40), we have

$$\mu = \frac{\delta^Q}{\gamma} Q. \qquad (52)$$

Substituting (52) into (49) and using $\bar{\mu} = \eta Q$, $L^H = \chi \tilde{H}(\varphi, Q)$ and (46) we obtain

$$\frac{\delta^Q}{\gamma} Q = \left(\frac{\frac{1}{Q} - \eta}{\frac{\delta^Q}{\gamma} - \eta} \right)^{\frac{\vartheta}{1-\vartheta}} \xi^{\frac{1}{1-\vartheta}} (\Gamma - 1)(1 + \bar{r}) \chi \tilde{H}(\varphi, Q) - \frac{\bar{r} - g}{1 + g}, \qquad (53)$$

which implicitly defines $\hat{Q}$. We see that $\hat{Q}$ is unique when $\tilde{H}(\varphi, Q)$ is non-increasing in Q. The other long run values follow.

B. Calibration (Positive Analysis)

We first consider the output elasticity of labor, $1 - \alpha = wL^Y/Y$. According to Karabarbounis and Neiman (2014, "CLS KN merged"), the arithmetic average for the period 1987-2011 of the UK corporate labor share in total income has been 62 percent (which is also the 2011 value). Thus, we set $\alpha = 0.38$. For the real interest rate we choose the typical value $\bar{r} = 0.05$. For the depreciation rate of physical capital we follow Grossmann and Steger (2016) who argue that $\delta^K = 0.07$. The growth rate of wage rates is set equal to the annual growth rate of income per capita in the UK for period 1960-2011, $g = 0.02$ (Jones, 2016b).

We assume that individuals become economically active at age 20 and live for a

maximum of 100 additional years (i.e., nobody reaches age 121); thus, $T = 101$. In fact, for modern times, 120 years seems to be the maximum life-span, irrespective of increasing life-expectancy in the last decades. The retirement age is reached after $R = 43$ working years (i.e. at age 63).[31]

We set $d_{\min}$ equal to the average health deficit index for a 20 year old in recent times. Using Canadian data, Mitnitski et al. (2002a) suggest $d_{\min} = 0.03$. Empirical evidence also suggests that the deficit state that leads to death for sure approximately is about two thirds (e.g. Harttgen et al., 2013); thus, $d_{\max} = 0.67$.

The remaining parameters are the mortality rate curvature parameters (σ, ϕ), the labor requirement per unit of health good (χ), medical R&D technology parameters $(\xi, \delta^Q, \vartheta)$, innovation step size (γ), the strength of the intertemporal innovation spillover (η), the price mark-up (Γ), health deficit accumulation parameters (ϱ, κ), the initial quality index of health goods (Q_0), the time path of the health care wedge, $\{\varphi_t\}_{t=0}^{\infty}$, the time path of initial survival rates, $\{S_{v,v}\}_{v=0}^{\infty}$, and health deficits of all cohorts with living members in the initial period (denoted by vector $\boldsymbol{d}_0$).[32] They are chosen to simultaneously match the observables highlighted in the main text: (i) empirical UK survival rates for ages 20-100 and periods 1950, 1970, 1990, 2010, (ii) the UK ratio of health expenditure to GDP $(\mathfrak{s}_t)$ between 1980-2010, (iii) the recent average rate of change of the health deficit index $(d_{v,t})$ in the cross-section of Canadian cohorts, (iv) the UK employment share in the health sector (ℓ_t^H). We assume that changes in the health care wedge are anticipated by economic subjects for the baseline calibration.

To match UK survival rates from year 1950 onwards (www.mortality.org), as $T = 101$, we need to specify initial conditions for the deficit index of all cohorts with living members in year 1850, i.e. we choose 1850 as initial period $(t = 0)$. Suppose initial deficit states, $\boldsymbol{d}_0$, result from a policy regime in which a health care system has never existed (i.e.

---

[31]In the UK, the average age of withdrawl from the labor market is around 64 for males and slightly below 62 for females in the 2000s (Mitchell and Guled, 2010).

[32]Initial labor productivity, A_0, does not enter the dynamical system for the positive analysis (Appendix A).

$\varphi_0 = 1$).[33] According to (1), (2) and (15), given $\boldsymbol{d}_0$ and $d_{\min}$, the evolution of survival functions is exclusively driven by the exogenous time paths $\{\varphi_t\}_{t=0}^{\infty}$ and $\{S_{v,v}\}_{v=0}^{\infty}$, and the endogenous time path of medical knowledge, $\{Q_t\}_{t=0}^{\infty}$. Matching them turns out to require that φ_t is gradually declining and $S_{v,v}$ is non-decreasing over time.[34] The assumed time path of $S_{v,v}$ is plausible as it reflects decreases over time in both child mortality and fatal accidents for young individuals. The assumed time path of φ_t is roughly consistent with the historical improvements in the British health care system (e.g. Stewart, 2015).[35] Despite the trend of increased health care access over time, advanced country health systems are at present still characterized by under-utilization in various forms. First, the density of physicians is much lower in rural areas than in urban areas, suggesting that access to health care is limited in rural regions (OECD, 2015, Fig. 7.10). Second, there is health care rationing, for instance, through waiting lists for orthopedic surgeries and other forms (OECD, 2015, Fig. 7.11-7.13). Some rationing measures have been introduced in the UK only recently (Edwards et al., 2015). We assume for the baseline calibration that, nevertheless, φ_t moderately decreases from about 0.15 in 2010 to 0.05 in year 2080. The initial quality index of health goods (in 1870), Q_0, is one percent of the steady state value of Q that results for $\varphi = 0.05$.[36]

A steady state analysis is instructive to understand the relationship between endogenous observables and helps us to calibrate the model. First, setting $Q_{t+1} = Q_t = Q$ in (41) and using both $\bar{\mu} = \eta Q$ and $\tilde{\mu} = \xi(L^Q)^{1-\vartheta}$, we obtain

$$[\bar{\mu} + (1 - \bar{\mu})\,\tilde{\mu} =]\,\mu = \frac{\delta^Q}{\gamma\eta}\bar{\mu} = \frac{\delta^Q}{\gamma}Q. \tag{54}$$

---

[33]Formally, denote by $\mathfrak{d}_{\mathfrak{a},0}$ the deficit index of a surviving individual of age $\mathfrak{a}$ in period 0. According to (15), we have $\mathfrak{d}_{\mathfrak{a},0} = d_{\min}(1+\varrho)^{\mathfrak{a}}$ for all $\mathfrak{a} \in [0, \bar{\mathfrak{a}}_0]$, where $\bar{\mathfrak{a}}_0$ is the maximum age in period 0. Thus, $\boldsymbol{d}_0 = (\mathfrak{d}_{1,0}, \mathfrak{d}_{2,0}, \mathfrak{d}_{3,0}, ..., \mathfrak{d}_{\bar{\mathfrak{a}}_0,0})$.

[34]Figure A.2 in Online-Appendix shows the exact time paths for the baseline calibration.

[35]For the past, innovations associated with health improvements may not exclusively be interpreted as being associated with the health sector but include better sanitation and better environmental conditions that more individuals received access to over time.

[36]Our calibrated model leads to the case where steady state quality of health goods $\hat{Q} \equiv \lim_{t\to\infty} Q_t < \bar{Q}_t$. We can verify that the steady state equilibrium of the calibrated model is saddle-point stable.

Thus, in the long run, the total innovation probability μ is proportional to $\bar{\mu}$ and thus proportional to the medical knowledge stock, Q. Second, according to (40),

$$\frac{(L^Q)^\vartheta}{\xi} = \frac{(1 - \bar{\mu})L^Q}{\mu - \bar{\mu}}. \tag{55}$$

Combining (55) with (49) and using both $L^H = \chi H$ and (54) implies that

$$\ell^Q = \frac{\frac{\delta^Q}{\gamma\eta} - 1}{\frac{1}{\bar{\mu}} - 1} \frac{(\Gamma - 1)(1 + \bar{r})\ell^H}{\frac{\bar{r} - g}{1 + g} + \frac{\delta^Q}{\gamma\eta}} \tag{56}$$

holds in the long run (recall that $\delta^Q > \gamma\eta$). Third, according to (20), the health expenditure share can be written as

$$\mathfrak{s} = \frac{pH}{Y + pH} = \frac{1}{\frac{Y}{pH} + 1} = \frac{1}{\frac{L^Y}{\Gamma\omega L^H}\left(\frac{K^Y}{AL^Y}\right)^\alpha + 1} = \frac{1}{\frac{\ell^Y}{(1-\alpha)\Gamma\ell^H} + 1}, \tag{57}$$

where we used (3) and (12) for the third equation and $\omega = (1 - \alpha)(AL^Y/K^Y)^{-\alpha}$ for the final one.

Unfortunately, we do not have data for the UK employment share of medical R&D workers (ℓ_t^Q). It is critically determined by the price mark up for medical goods (Γ). For $\Gamma = 1.25$, we obtain $\ell_t^Q = 0.012$ for the year 2010.[37] Moreover, the calibrated model implies a non-profit driven and total innovation probability of 4.1 and 9 percent, respectively, ($\bar{\mu}_{t+1} = 0.034$, $\mu_{t+1} = 0.08$). The implied effective patent length (EPL), i.e. the inverse of the probability of an incumbent to be driven out of the market is the effective patent length, is given by $EPL_t \equiv 1/\mu_{t+1} = 12.5$. It is close to the 10 years

---

[37]The implied share of workers in medical R&D occupations of about one percent seems high at the first glance, if a strict definition of this occupation is applied, like medical scientists and engineers. For instance, we may consider US data from the Bureau of Labor Statistics (2016) for data availability reasons. Adding up for the year 2015 the number of biological scientists (102,000 employees), medical scientists (110,000 employees) and biomedical engineers (21,000 employees) suggests a combined employment share of only 1.7 per mill. However, a more appropriate interpretation of employment related endogenous technical progress in our model requires to add managers (for strategic decisions and marketing) and other professionals (like patent lawyers) who organize and commercialize medical R&D.

assumed in Jones and Williams (2000). Finally, the ratio of population size aged 63+ (retirement age) to the population size aged 20-62 (working age), DPR_t, implied by the calibrated model is 40 percent for 2010.[38]

C. Consumption Paths (Normative Analysis)

- **Anticipated Health Policy:** Let us start with the case without unanticipated policy shocks. Using $S_{v,t} = S_{v,v} \prod_{u=v}^{t-1}(1 - m_{v,u})$ in (22), the Lagrangian $\mathcal{L}_v$ associated with maximizing U_v subject to (25) and $a_{v,v+T} \geq 0$ is

$$
\begin{aligned}
\mathcal{L}_v \;=\; & ... + \beta^{t-v} S_{v,v} \prod_{u=v}^{t-1}(1 - m_{v,u}) \frac{\log c_{v,t}}{(1 + d_{v,t})^{\zeta}} + \\
& \beta^{t+1-v} S_{v,v} \prod_{u=v}^{t}(1 - m_{v,u}) \frac{\log c_{v,t+1}}{(1 + d_{v,t+1})^{\zeta}} + ... + \\
& \lambda_{v,t}\left[(1 - \tau_t)w_t + (1 + r_{v,t})a_{v,t} - c_{v,t} - a_{v,t+1}\right] + \\
& \lambda_{v,t+1}\left[(1 - \tau_{t+1})w_{t+1} + (1 + r_{v,t+1})a_{v,t+1} - c_{v,t+1} - a_{v,t+2}\right] + ... \quad (58)
\end{aligned}
$$

where $\lambda_{v,t}$, $\lambda_{v,t+1}$, etc. denote the multipliers for period t, $t+1$, etc. The first-order conditions $\partial \mathcal{L}_v / \partial c_{v,t} = \partial \mathcal{L}_v / \partial c_{v,t+1} = \partial \mathcal{L}_v / \partial a_{v,t+1} = 0$ can be written as

$$
\frac{\beta^{t-v} S_{v,v} \prod_{u=v}^{t-1}(1 - m_{v,u})}{(1 + d_{v,t})^{\zeta} c_{v,t}} = \lambda_{v,t}, \tag{59}
$$

$$
\frac{\beta^{t+1-v} S_{v,v} \prod_{u=v}^{t}(1 - m_{v,u})}{(1 + d_{v,t+1})^{\zeta} c_{v,t+1}} = \lambda_{v,t+1}, \tag{60}
$$

$$
\lambda_{v,t} = \lambda_{v,t+1}(1 + r_{v,t+1}). \tag{61}
$$

Combining (59)-(61) leads to

$$
\frac{(1 + d_{v,t+1})^{\zeta} c_{v,t+1}}{(1 + d_{v,t})^{\zeta} c_{v,t}} = \beta(1 - m_{v,t})(1 + r_{v,t+1}). \tag{62}
$$

---

[38]This is considerably higher than the level in the data (33.1 percent); see Office for National Statistics (2016). The deviation mainly reflects our neglect of recent immigration into the UK labor market that was primarily enabled by the free movement of labor within the European Union. In the next section, we will thus interpret the change of DPR_t over time rather than its level.

Using (26) in (62) implies

$$c_{v,t+1} = \left(\frac{1 + d_{v,t}}{1 + d_{v,t+1}}\right)^\zeta \beta(1 + \bar{r})c_{v,t}. \tag{63}$$

Iterating and using $d_{v,v} = d_{\min}$, we obtain

$$c_{v,t} = \left(\frac{1 + d_{\min}}{1 + d_{v,t}}\right)^\zeta \beta^{t-v}(1 + \bar{r})^{t-v}c_{v,v}. \tag{64}$$

From (25), (26), $a_{v,v} = 0$ and $a_{v,v+T} = 0$ (reflecting that it is optimal not to hold wealth after certain death), we find that the intertemporal budget constraint of a member of cohort v is given by

$$c_{v,v} + \sum_{t=v+1}^{v+T-1} \left(\frac{c_{v,t}}{\prod_{u=v+1}^{t}(1 + r_{v,u})}\right) = (1 - \tau_v)w_v + \sum_{t=v+1}^{v+R-1} \left(\frac{(1 - \tau_t)w_t}{\prod_{u=v+1}^{t}(1 + r_{v,u})}\right). \tag{65}$$

Using (26) and (64), we obtain for the left-hand side of (65) that

$$c_{v,v} + \sum_{t=v+1}^{v+T-1} \left(\frac{c_{v,t}}{\prod_{u=v+1}^{t}(1 + r_{v,u})}\right) = c_{v,v}\left(1 + \sum_{t=v+1}^{v+T-1} \beta^{t-v}\left(\frac{1 + d_{\min}}{1 + d_{v,t}}\right)^\zeta \prod_{u=v}^{t-1}(1 - m_{v,u})\right). \tag{66}$$

Equating the right-hand sides of (65) and (66), and using (26), $w_t = \omega A_t$ with ω given by (4), (2) with $S_{v,v} = 1$ and $A_t = A_v(1 + g)^{t-v}$, implies that the initial consumption level, $c_{v,v}$, is given by

$$c_{v,v} = \omega A_v \frac{1 - \tau_v + \sum_{t=v+1}^{v+R-1}(1 - \tau_t)\left(\frac{1+g}{1+\bar{r}}\right)^{t-v}\frac{S_{v,t}}{S_{v,v}}}{1 + \sum_{t=v+1}^{v+T-1} \beta^{t-v}\left(\frac{1+d_{\min}}{1+d_{v,t}}\right)^\zeta \frac{S_{v,t}}{S_{v,v}}}. \tag{67}$$

- **Unanticipated Health Policy Shock:** We now turn to the case where some individuals experience an unanticipated policy shock in period t_0. That is, for $t < t_0$ they follow the same consumption path as computed in the previous case and then they re-optimize in t_0. According to (63), knowing c_{v,t_0}, the path of consumption

43

of any living member of generation v for future dates for $t \geq t_0$ evolves as

$$c_{v,t} = \left(\frac{1 + d_{v,t_0}}{1 + d_{v,t}} \right)^\zeta \beta^{t-t_0} \left(1 + \bar{r} \right)^{t-t_0} c_{v,t_0}. \tag{68}$$

Using (25) and $a_{v,v} = 0$, for $t_0 < v + R$ we have

$$\frac{a_{v,t_0}}{\prod_{u=v+1}^{t_0-1} \left(1 + r_{v,u} \right)} = \left(1 - \tau_v \right) w_v - c_{v,v} + \sum_{t=v+1}^{t_0-1} \frac{\left(1 - \tau_t \right) w_t - c_{v,t}}{\prod_{u=v+1}^{t} \left(1 + r_{v,u} \right)} \tag{69}$$

Using (26), (64) and (2), we obtain

$$c_{v,v} + \sum_{t=v+1}^{t_0-1} \left(\frac{c_{v,t}}{\prod_{u=v+1}^{t}(1 + r_{v,u})} \right) = c_{v,v} \left(1 + \sum_{t=v+1}^{t_0-1} \beta^{t-v} \left(\frac{1 + d_{\min}}{1 + d_{v,t}} \right)^\zeta \frac{S_{v,t}}{S_{v,v}} \right). \tag{70}$$

Using (26) and (2), we also get

$$\frac{1}{\prod_{u=v+1}^{t_0-1} \left(1 + r_{v,u} \right)} = \frac{S_{v,t_0-1}}{S_{v,v}(1 + \bar{r})^{t_0-1-v}}. \tag{71}$$

Substituting (70), (71), $w_t = \omega A_t$ and $A_t = A_v(1 + g)^{t-v}$ into (69), the wealth holding of a member of generation v in $t_0 < v + R$ is given by

$$\begin{aligned} a_{v,t_0} &= \left(1 + \bar{r} \right)^{t_0-v-1} \frac{S_{v,v}}{S_{v,t_0-1}} \left[A_v \omega \left(1 - \tau_v + \sum_{t=v+1}^{t_0-1} \left(1 - \tau_t \right) \left(\frac{1 + g}{1 + \bar{r}} \right)^{t-v} \frac{S_{v,t}}{S_{v,v}} \right) - \right. \\ & \left. c_{v,v} \sum_{t=v+1}^{t_0-1} \left(\frac{1 + d_{\min}}{1 + d_{v,t}} \right)^\zeta \beta^{t-v} \frac{S_{v,t}}{S_{v,v}} - c_{v,v} \right]. \end{aligned} \tag{72}$$

Analogously, for $t_0 \geq v + R$, we have

$$\begin{aligned} a_{v,t_0} &= \left(1 + \bar{r} \right)^{t_0-v-1} \frac{S_{v,v}}{S_{v,t_0-1}} \left[A_v \omega \left(1 - \tau_v + \sum_{t=v+1}^{v+R-1} \left(1 - \tau_t \right) \left(\frac{1 + g}{1 + \bar{r}} \right)^{t-v} \frac{S_{v,t}}{S_{v,v}} \right) - \right. \\ & \left. c_{v,v} \sum_{t=v+1}^{t_0-1} \left(\frac{1 + d_{\min}}{1 + d_{v,t}} \right)^\zeta \beta^{t-v} \frac{S_{v,t}}{S_{v,v}} - c_{v,v} \right]. \end{aligned} \tag{73}$$

Recall that $c_{v,v}$ is the initial consumption level chosen before the unanticipated shock occurs. Next, use (25) and $a_{v,v+T} = 0$ to obtain

$$c_{v,t_0} + \sum_{t=t_0+1}^{v+T-1} \frac{c_{v,t}}{\prod_{u=t_0+1}^{t}(1+r_{v,u})} = (1+r_{v,t_0})a_{v,t_0} + (1-\tau_{t_0})w_{t_0} + \sum_{t=t_0+1}^{v+R-1} \frac{(1-\tau_t)w_t}{\prod_{u=t_0+1}^{t}(1+r_{v,u})}. \tag{74}$$

Using (68) implies

$$c_{v,t_0} + \sum_{t=t_0+1}^{v+T-1} \frac{c_{v,t}}{\prod_{u=t_0+1}^{t}(1+r_{v,u})} = c_{v,t_0}\left(1 + \sum_{t=t_0+1}^{v+T-1}\left(\frac{1+d_{v,t_0}}{1+d_{v,t}}\right)^\zeta \beta^{t-t_0} \prod_{u=t_0}^{t-1}(1-m_{v,u})\right). \tag{75}$$

Equating the right-hand sides of (74) and (75) and using (26), $w_t = \omega A_t$ and $A_t = A_{t_0}(1+g)^{t-t_0}$ implies, for $t_0 < v + R$, the consumption level:

$$c_{v,t_0} = \frac{\frac{1+\bar{r}}{1-m_{v,t_0-1}}a_{v,t_0} + \omega A_{t_0}\left(1 - \tau_{t_0} + \sum_{t=t_0+1}^{v+R-1}(1-\tau_t)\left(\frac{1+g}{1+\bar{r}}\right)^{t-t_0}\prod_{u=t_0}^{t-1}(1-m_{v,u})\right)}{1 + \sum_{t=t_0+1}^{v+T-1}\left(\frac{1+d_{v,t_0}}{1+d_{v,t}}\right)^\zeta \beta^{t-t_0}\prod_{u=t_0}^{t-1}(1-m_{v,u})} \tag{76}$$

with a_{v,t_0} given by (72) and $A_{t_0} = A_v(1+g)^{t_0-v}$. Analogously, for $t_0 \geq v + R$ (i.e. the individual is retired when the shock hits), we have

$$c_{v,t_0} = \frac{\frac{1+\bar{r}}{1-m_{v,t_0-1}}a_{v,t_0}}{1 + \sum_{t=t_0+1}^{v+T-1}\left(\frac{1+d_{v,t_0}}{1+d_{v,t}}\right)^\zeta \beta^{t-t_0}\prod_{u=t_0}^{t-1}(1-m_{v,u})} \tag{77}$$

with a_{v,t_0} given by (73).

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Online-Appendix:
Additional Figures and Tables

List of 40 Variables included in the frailty index	Cut Point
Help Bathing	Yes = 1, No = 0
Help Dressing	Yes = 1, No = 0
Help getting in/out of Chair	Yes = 1, No = 0
Help Walking around house	Yes = 1, No = 0
Help Eating	Yes = 1, No = 0
Help Grooming	Yes = 1, No = 0
Help Using Toilet	Yes = 1, No = 0
Help up/down Stairs	Yes = 1, No = 0
Help lifting 10 lbs	Yes = 1, No = 0
Help Shopping	Yes = 1, No = 0
Help with Housework	Yes = 1, No = 0
Help with meal Preparations	Yes = 1, No = 0
Help taking Medication	Yes = 1, No = 0
Help with Finances	Yes = 1, No = 0
Lost more than 10 lbs in last year	Yes = 1, No = 0
Self Rating of Health	Poor = 1, Fair = 0.75, Good = 0.5, V. Good = 0.25, Excellent = 0
How Health has changed in last year	Worse = 1, Better/Same = 0
Stayed in Bed at least half the day due to health (in last month)	Yes = 1, No = 0
Cut down on Usual Activity (in last month)	Yes = 1, No = 0
Walk outside	<3 days = 1, ≤ 3 days = 0
Feel Everything is an Effort	Most of time = 1, Some time = 0.5, Rarely = 0
Feel Depressed	Most of time = 1, Some time = 0.5, Rarely = 0
Feel Happy	Most of time = 0, Some time = 0.5, Rarely = 1
Feel Lonely	Most of time = 1, Some time = 0.5, Rarely = 0
Have Trouble getting going	Most of time = 1, Some time = 0.5, Rarely = 0
High blood pressure	Yes = 1, Suspect = 0.5, No = 0
Heart attack	Yes = 1, Suspect = 0.5, No = 0
CHF	Yes = 1, Suspect = 0.5, No = 0
Stroke	Yes = 1, Suspect = 0.5, No = 0
Cancer	Yes = 1, Suspect = 0.5, No = 0
Diabetes	Yes = 1, Suspect = 0.5, No = 0
Arthritis	Yes = 1, Suspect = 0.5, No = 0
Chronic Lung Disease	Yes = 1, Suspect = 0.5, No = 0
MMSE	<10 = 1, 11–17 = 0.75, 18–20 = 0.5, 20–24 = 0.25, >24 = 0
Peak Flow	See Table 2
Shoulder Strength	See Table 2
BMI	See Table 2
Grip Strength	See Table 2
Usual Pace	See Table 2
Rapid Pace	See Table 2

Variable	Deficit for Men	Deficit for Women
Peak Flow (liters/min)	≤ 340	≤ 310
Body Mass Index (BMI)	<18.5, ≥ 30 as a deficit. 25-<30 as a 'half deficit'	<18.5, ≥ 30 as a deficit. 25-<30 as a 'half deficit'
Shoulder Strength (kg)	≤ 12	≤ 9
Grip Strength (GS in kg)	For BMI ≤ 24, GS ≤ 29 For BMI 24.1–28, GS ≤ 30 For BMI >28, GS ≤ 32	For BMI ≤ 23, GS ≤ 17 For BMI 23.1–26, GS ≤ 17.3 For BMI 26.1–29, GS ≤ 18 For BMI>29, GS ≤ 21
Rapid pace Walk (sec)	>10	>10
Usual pace Walk (sec)	>16	>16

Table A.1: List of deficits in Searle et al. (2008, Tab. 1 and 2)

Note: The individual health deficit index in Searle et al. (2008) is computed by summing up the cut points for an individual and dividing by 40.
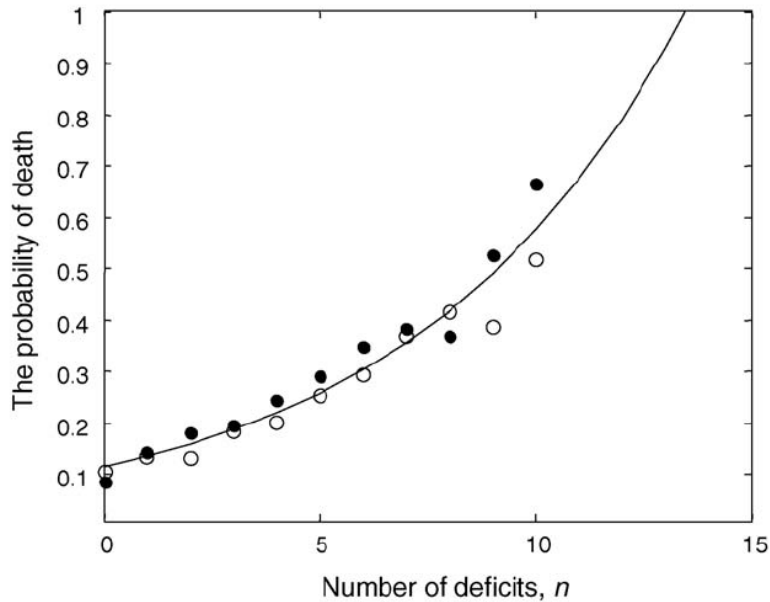
Figure A.1: Mortality rates and the number of health deficits (out of 31 potential deficits) for a cross section of Canadian cohorts aged 65+ from three waves.

Source: Mitnitski, Bao and Rockwood (2006, Fig. 2).

Notes: (1) Data from the Canadian Study of Health and Aging (CSHA), "a representative cohort study designed to study dementia and other age-related problems [...]. Briefly, in 1990-1991, during the first wave of the study (CSHA-1) 9008 community-dwelling people age 65 and over were assessed using a self-report questionnaire, of whom complete data are available for 5586 survivors for the second wave (CSHA-2, conducted in 1995-1996) and 3211 for the third wave (CSHA-3, conducted in 2000-2001)." Mitnitski et al. (2006, p. 492). (2) Original note: "Probability estimates come from the combined model of CSHA-1 to CSHA-2 (filled circles), and CSHA-2 to CSHA-3 (empty circles). Circles represent observational data and lines show the fit."
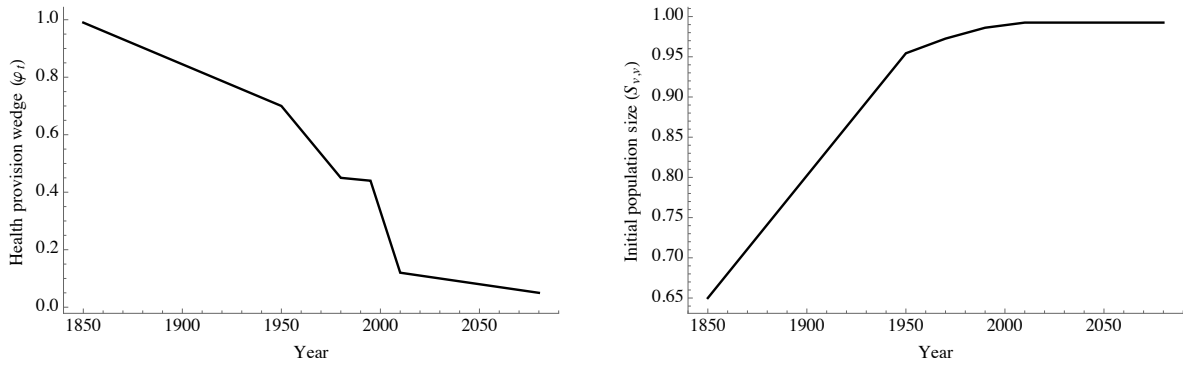
Figure A.2: Calibration of the time paths of the health care wedge and initial cohort sizes in the baseline scenario.
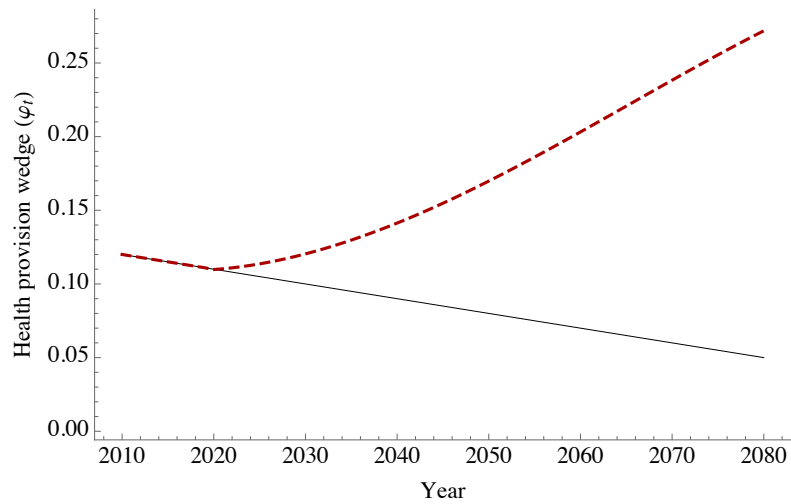

Figure A.3: Calibration of the time paths of the health care wedge from year 2010 onwards in the reform scenario (dashed line) and baseline scenario (thin line).

	1950		1970		1990		2010	
Age	Data	Model	Data	Model	Data	Model	Data	Model
20	51.8	53.0	53.8	54.4	56.7	56.8	61.0	60.6
25	47.0	48.1	49.0	49.4	51.9	51.9	56.1	55.7
30	42.4	43.2	44.2	44.6	47.1	47.0	51.2	50.8
35	37.8	38.4	39.4	39.8	42.3	42.2	46.4	46.0
40	33.2	33.7	34.7	35.0	37.5	37.5	41.6	41.2
45	28.7	29.1	30.1	30.4	32.8	32.8	36.9	36.5
50	24.4	24.7	25.7	26.0	28.2	28.3	32.3	31.9
55	20.3	20.5	21.6	21.7	23.9	24.0	27.8	27.5
60	16.6	16.6	17.7	17.7	19.8	19.9	23.5	23.3
65	13.2	13.0	14.3	14.1	16.1	16.1	19.4	19.3
70	10.2	9.9	11.3	10.8	12.9	12.6	15.5	15.6
75	7.6	7.2	8.7	8.0	10.0	9.6	12.0	12.2
80	5.6	5.0	6.5	5.7	7.5	7.0	8.9	9.3
85	4.1	3.4	4.8	3.9	5.5	4.9	6.4	6.8
90	3.0	2.1	3.5	2.5	4.0	3.3	4.4	4.8
95	2.2	1.2	2.6	1.5	2.9	2.1	3.1	3.2
100	1.8	0.6	2.0	0.8	2.1	1.2	2.2	2.0

Table A.2: Comparison of remaining period life expectancy according to age: UK data vs calibrated model, for years 1950, 1970, 1990, 2010.

Age	2020 Baseline	2050 Baseline	2050 Reform	2080 Baseline	2080 Reform
20	63.6	73.5	72.7	83.7	79.1
25	58.7	68.6	67.9	78.8	74.3
30	53.8	63.8	63.0	74.0	69.4
35	49.0	59.0	58.2	69.2	64.6
40	44.2	54.2	53.4	64.4	59.9
45	39.5	49.4	48.7	59.6	55.2
50	34.8	44.8	44.0	54.9	50.5
55	30.3	40.1	39.4	50.3	46.0
60	26.0	35.6	34.9	45.7	41.5
65	21.9	31.2	30.5	41.2	37.2
70	18.0	26.9	26.3	36.8	33.0
75	14.4	22.9	22.3	32.5	28.9
80	11.2	19.0	18.5	28.4	25.0
85	8.5	15.5	15.0	24.4	21.3
90	6.1	12.3	11.9	20.5	17.8
95	4.3	9.5	9.1	16.9	14.5
100	2.8	7.1	6.8	13.4	11.4

Table A.3: Implied remaining period life expectancies according to age: baseline vs. reform scenario for years 2020, 2050, 2080.

Age	2020 Baseline	2050 Baseline	2050 Reform	2080 Baseline	2080 Reform
20	86.2	91.0	80.6	92.8	80.6
25	80.2	85.5	75.8	87.5	75.7
30	74.2	80.0	71.0	82.3	70.8
35	68.0	74.5	66.2	77.0	66.1
40	61.4	68.9	61.4	71.7	61.4
45	54.6	63.4	56.6	66.4	56.7
50	47.8	57.8	51.8	61.1	52.1
55	41.1	52.2	47.0	55.8	47.6
60	34.7	46.7	42.3	50.5	43.1
65	28.7	41.1	37.4	45.3	38.7
70	23.1	35.5	32.3	40.2	34.4
75	18.1	29.8	27.3	35.2	30.2
80	13.7	24.4	22.4	30.3	26.1
85	10.0	19.5	18.0	25.5	22.0
90	7.0	15.2	14.0	20.9	18.0
95	4.7	11.4	10.5	16.5	14.2
100	3.0	8.2	7.6	12.6	10.8

Table A.4: Implied remaining cohort life expectancies according to age: baseline vs. reform scenario for years 2020, 2050, 2080.